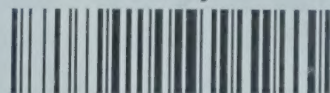


**NAUTRITIONAL DISEASES
IN INDIA**

R. PASSMORE

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NUTRITIONAL DISEASES IN INDIA

DESCRIBED FOR STUDENTS AND PRACTITIONERS

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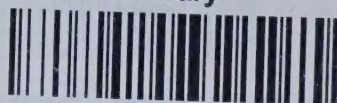
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PREFACE

Considering the number of excellent books on nutrition, which are available, the addition of an extra volume calls for an explanation. This book aims at providing a practical account of nutritional diseases in India, suitable for medical students during their clinical period and for general practitioners in India. It makes no pretence to be a complete monograph on the subject. Several excellent large reference works already exist for the use of research workers and senior physicians. Neither is it in any sense a popular book for the general public. There is already in existence a large collection of books, written both in India and in England, in which the educated housewife and the man-in-the-street can find the latest accounts of nutritional science and their application to practical dietetics. However the ordinary doctor and medical student, who have to rely on the standard text-books of Medicine and Tropical Medicine in use in the Medical Schools and Colleges in India, find within them accounts of nutritional diseases, which differ in many important aspects from the manner, in which these diseases present themselves to the practitioner in India.

In this book the main object is to describe the signs and symptoms of malnutrition and deficiency diseases, so that these conditions can be readily and accurately diagnosed. Accepted and practical methods of treatment are given in detail. Throughout the public health importance of nutrition is stressed and the relevant epidemiological factors and their relation to social conditions described and discussed.

In writing for the student, the difficult problem is to judge what part of the subject can with reason be omitted. The bulk of medical knowledge is now so vast and the weight of text-books, which the student is expected to read, increases so rapidly that it becomes more and more difficult for him to assimilate and digest what he reads. The amount of factual knowledge, which has to be absorbed, is in danger of becoming so extensive as to preclude any time being left available for pondering on its meaning and significance. The work of the modern medical student is so great, that every effort must be made to spare him the burden of unnecessary learning. Thus the writer for the student must resist the temptation to include the latest and perhaps most intriguing speculations of research workers on small, even if important, points: the rare and the obscure, however interesting, must be excluded and the essential features of the subject presented as they appear in the everyday practice of medicine. The writer must be ready to suffer the learned critics, who point out the number and extent of his omissions.

In this book the chemical aspects of the subject have been cut down to a minimum. Clinical, as opposed to laboratory, diagnosis has been stressed throughout. There is little or no discussion on the exact role of each recently

discovered vitamin in particular syndromes. The emphasis is laid throughout on food deficiencies rather than on vitamin deficiencies. The pathological aspects of nutritional diseases, often a difficult and controversial subject, have purposely been reduced to a minimum. By these means more space is available for discussing the associated public health and social problems, with which every doctor in India must be familiar. Social medicine has recently emerged as a separate academic discipline in certain medical colleges. In no country in the world is there a greater need for its study than in India or more scope for its practical applications.

No account of the elementary principles of nutrition is given and a knowledge of physiology up to accepted M. B. standards is assumed. However a brief summary of the properties of the important vitamins is given in an appendix, since knowledge of these substances is extending so rapidly.

In compiling a book of this nature, frequent reference has naturally been made to the writings of other workers, mostly in technical journals. In some places an apology may be necessary for omitting to acknowledge this debt to others. However it was thought to be both unnecessary and unwise to interrupt the text of a book for students with references to original papers. Nevertheless a few key papers are listed at the end of each section or chapter: from these it will be possible for advanced students to get a complete guide to the literature of the subject.

The writer has had some ten years' experience of nutrition and its relation to diseases in India, before the war on the staff on the Indian Research Fund Association Laboratories, at Coonoor, and during the war as a physician on active service with the Indian Army. Much has been learnt from discussions with others, similarly interested in the subject. A great debt for assistance in the formation of ideas and in the collection of material is owed to numerous other medical officers, who served in the Indian army during the war, and also to Dr. Aykroyd, formerly the Director, and other members of the Coonoor staff.

R. PASSMORE.

COONOR.

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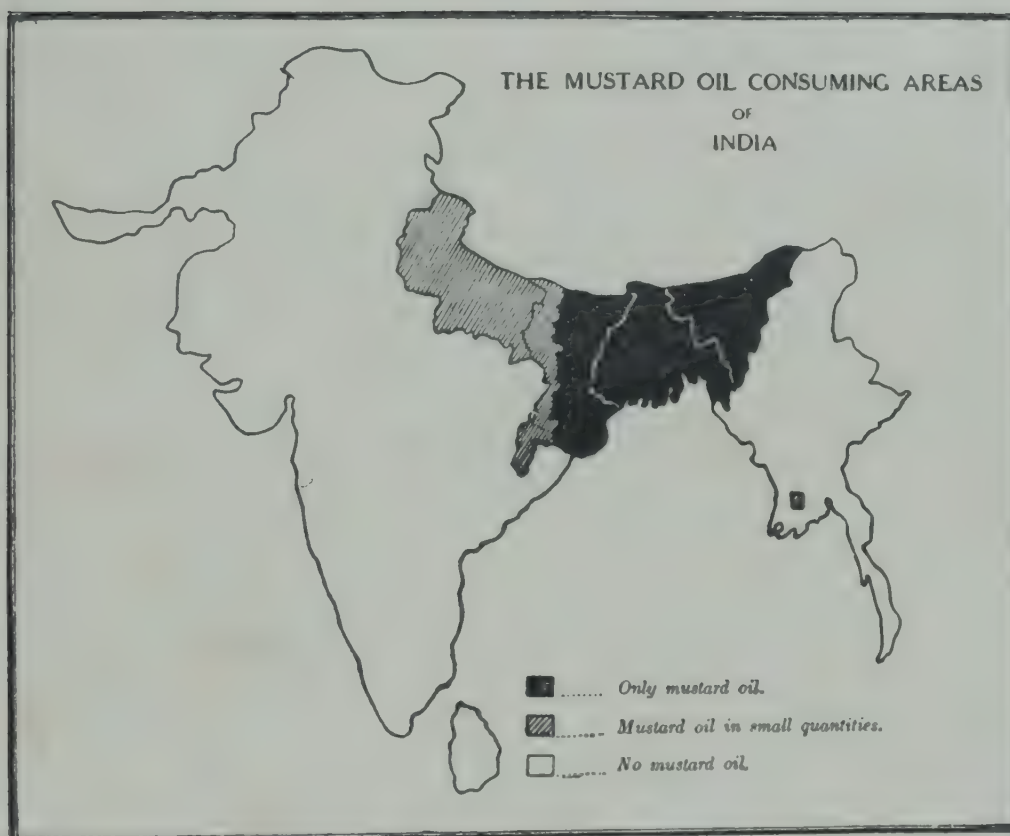
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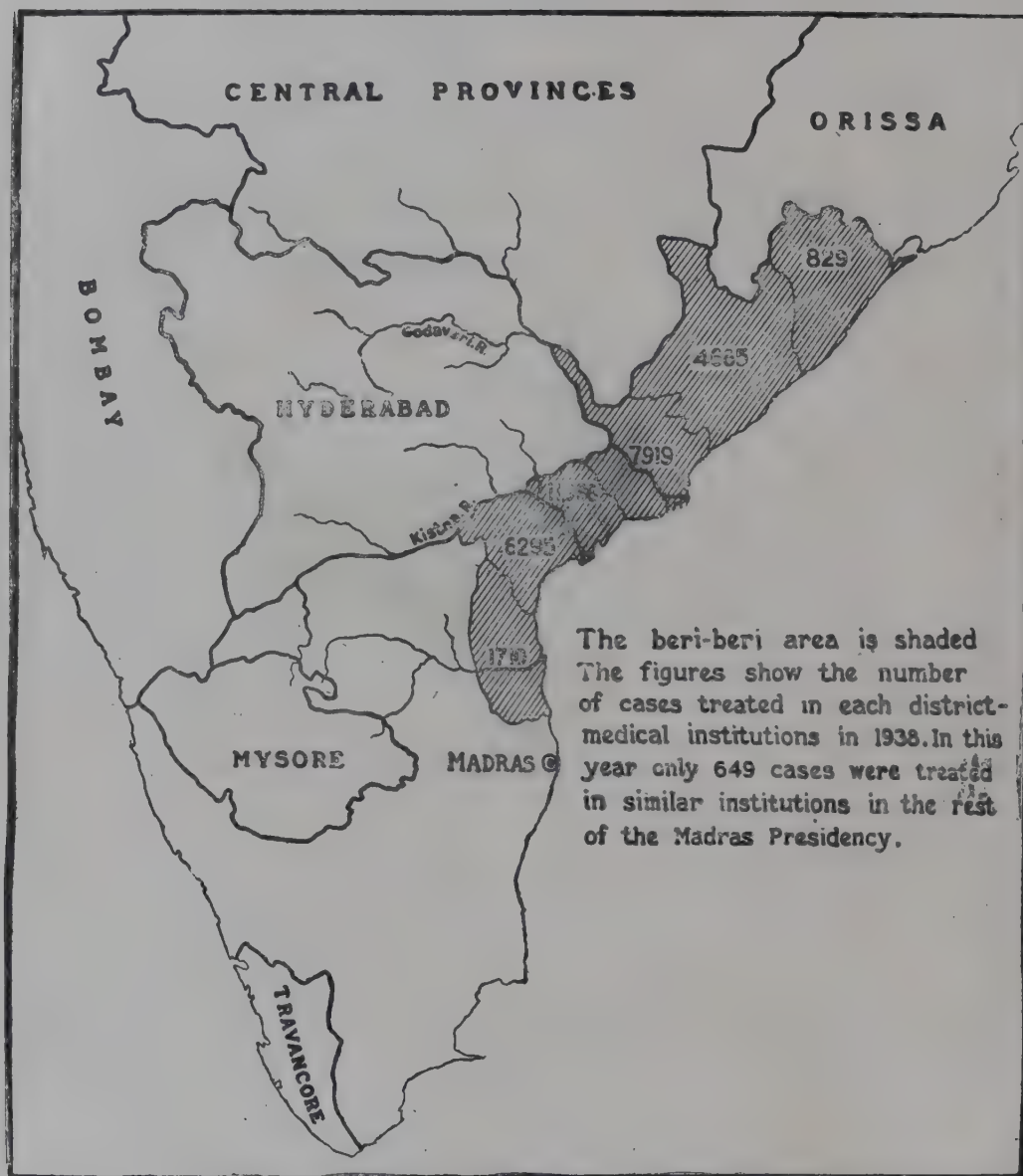
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Map 1.

The mustard oil consuming areas of India.
From R. B. Lall & S. C. Roy, (1937).
Indian Journal of Medical Research 25, 177.



Map 2.

Distribution of beriberi in the Madras Presidency.

NUTRITIONAL DISEASES IN INDIA

CHAPTER 1

THE PRINCIPAL CAUSES OF SICKNESS AND DEATH IN INDIA AND THEIR RELATION TO DIET

INTRODUCTION

“No preventive campaign against malaria, against tuberculosis or against leprosy, no maternity relief or child welfare activities are likely to achieve any great success unless those responsible recognise the vital importance of this factor of defective nutrition and from the very start give it their most serious attention. Abundant supplies of quinine and the multiplication of tuberculosis hospitals, sanatoria, leprosy colonies and maternity and child welfare centres are no doubt desirable, if not essential, but none of these go to the root of the matter. The first essentials for the prevention of disease are a higher standard of health, a better physique and a greater power of resistance to infection. These can only be obtained if the food of the people is such as will give all the physiological and nutritional requirements of the human frame.” *Report of the Public Health Commissioner with the Government of India, 1935.*

All experienced medical men in India will agree with this statement. Indeed similar ideas have frequently been expressed by doctors in all parts of India, although rarely so concisely as by Sir Alexander Russell in the above report. The effects on health of an insufficiency of food are most frequently indirect. Except during a great famine, deaths in India due to starvation are rare. Although there are numerous signs of ill health and disease amongst the people of India directly attributable to a shortage of essential articles of food, the most important effects of such a shortage is the reduction of the resistance of the people to the common infectious diseases. Many diseases can only flourish amongst a population, broken by poverty and malnutrition. Hence before describing the direct manifestations of dietary deficiencies in the human body, it is proper to consider first the chief causes of sickness and death in India and the manner in which improper feeding contributes to their spread.

Statisticians can calculate from the figures of the yearly number of births and deaths in India, how long on an average a baby born in India will live. This figure is known as the expectation of life. For India it is 26 years, a very low figure compared with countries such as England with 59, Sweden with 62 and New Zealand, perhaps the healthiest of all, with 68 years. In India the incidence of death from various causes is higher than in most other countries of the world and there is no doubt that many of these deaths could

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be prevented. All deaths in India have to be notified to the Public Health authorities, but as the majority of persons die unattended by a doctor, accurate knowledge of the causes of death is not available. The notifying authority, often the village headman, is restricted to seven diagnoses. The average number of deaths from each cause for the period 1929 to 1938 is given in the Table 1.

TABLE 1

Causes of Death				Number of Deaths per year.	Death rate per thousand.
Cholera	..	...	...	190,039	0·7
Small-pox	...	...	...	70,563	0·3
Plague	...	...	...	40,287	0·15
Fevers	...	...	...	3,710,168	13·8
Diarrhoea and Dysentery	..	...	...	261,662	1·0
Respiratory Diseases	...	..	...	455,240	1·7
Other diseases	...	..	...	1,659,639	5·8
Total deaths				6,387,657	23·0

What proportion of these deaths have their roots in malnutrition? Such statements as, "Half the patients in this hospital would not be here, if they had had the right kind of food to eat", have frequently been made by hospital physicians in India. As a generalisation, it is probably not an exaggeration. Actually it is quite impossible to make any quantitative assessment of the importance of faulty and insufficient diet as a disease-producing factor in India. Some light may be thrown on the question of the magnitude of the numbers by a study of the available figures for morbidity and mortality. In the present chapter it is proposed to discuss the principal causes of sickness and death and their relation to diet.

Let us consider the causes of death in table 1. Firstly, there are the three great pestilences of India, cholera, small-pox and plague. There is no evidence that nutrition plays any important part in the direct defence of the body against the organisms responsible for these diseases. Rich and poor, the hungry and well-fed may be fatally struck down alike by these scourges, which are no respecters of persons and have frequently worked their havoc in the palaces and castles of the great. Although these pestilences are between them responsible for an average of just over three hundred thousand deaths per year, this is less than five per cent of the total deaths. These diseases are not, at the moment, the major causes of death in India. However anyone familiar with their ups and downs throughout the world during historical times and with the gigantic pandemics, which they have caused, decimating populations

in the past, can have no certain confidence in the future. Each of these diseases can be prevented by appropriate sanitary administrative measures, which are well-known and established. Yet even now the general lethargy and administrative inertia is such that 300,000 preventable deaths are allowed to occur a year. A lively and well nourished population would not tolerate this annual waste.

“FEVERS” AND MALARIA

The group “fevers” ranks as “captain of the men of death” in India. A high proportion of the deaths ascribed to this cause are due to malaria. It has been estimated that about a hundred million persons suffer from malaria every year and that the annual number of deaths due to this cause exceeds 1.5 millions. The remark of the Public Health Commissioner (1937) that “malaria constitutes the major public health problem in India, both from the point of morbidity and mortality” is fully justified.

Insufficient knowledge is at present available as to the nature and relative importance of conditions other than malaria falling into the heterogeneous group “fevers”. The figures shown in Table 2, taken from the Public Health Commissioner’s Report for 1936, illustrate an attempt to analyse the death rate from “fevers” in the Punjab and Bengal in 1935.

TABLE 2
Mortality from “fevers” in the Punjab and Bengal (1935).

Disease.	PUNJAB.		BENGAL.	
	Deaths reported.	Percentage of the total.	Deaths reported.	Percentage of the total.
Malaria	168,327	45.0	337,647	47.1
Kala-azar	...	...	21,161	3.0
Enteric fever	6,512	1.7	8,359	1.2
Measles	1,340	0.4	4,549	0.6
Influenza	47	0.01	2,502	0.3
Relapsing fever	48	0.01	7	0.0
Typhus fever	2	0.00	1,551	0.2
Cerebrospinal fever	91	0.02	910	0.1
Blackwater fever	...	...	115	0.01
Other fevers	197,850	52.80	339,341	47.40
	374,817		716,142	

No useful purpose would be served by speculation about the relation between the various conditions listed other than malaria and malnutrition. The large and obscure group "other fevers" contributed 52.8 and 47.4 per cent of the mortality ascribed to all fevers in the two provinces respectively. The death rate from fevers other than malaria tends to rise during famine and possibly the group includes diseases which are more prone to attack and kill the ill-nourished than the well-fed. In the present state of public health organisation in India, the investigation of such problems presents formidable difficulties.

Many malariologists have drawn attention to an association between malaria and economic stress, and the invariable accompaniment of the latter, malnutrition. Hackett (1937) says that "current feeling seems to hold that malaria infection can be so interwoven with under-nutrition and low standards of living that the complex must be tackled as a whole, any independent solution of the malaria problem being out of the question". The same worker, however, remarks that the absence of experimental data makes the discussion of these questions a mere battle of opinion.

Fulminant epidemics of malaria have often been associated with scarcity and famine. To give one example, in 1897 an epidemic of fever occurring in the famine districts in the Central Provinces was the subject of a special enquiry. A theory was advanced that the fever was of a "specially malignant type" but this was not supported by the majority of observers. "Almost all the medical officers employed agreed in holding that the fever was ordinary malaria fever, which, though it attacked all classes more or less, was specially fatal only in the case of those who had suffered from privation" (Indian Famine Commission, 1898). Severe epidemics of malaria associated with famine usually occur when the long delayed rains have arrived and the people are returning to their normal village occupations. The rainfall which occurs after a period of drought is often excessive, giving rise to floods, and this in itself usually creates circumstances favourable for the transmission of malaria. There are other epidemiological factors which may play a part in such outbreaks. The years of drought preceding an epidemic may so lower anopheline density and longevity that little or no malaria transmission takes place for several consecutive years. The absence of malaria transmission during such prolonged periods allows the immunity of the population to fall to a low level especially in the younger children, many of whom may never have been exposed to malarial infection. In malaria epidemics the mortality among children is often exceptionally severe and forms a high proportion of total mortality. Widespread destruction of cattle may result in the deviation of cattle-feeding anophelines to man.

In certain circumstances drought may lead to increased anopheline density, and if this occurs the height of the malaria epidemic may coincide

with the height of the famine. Under certain geographical conditions, e.g., in submontane areas in South India and Ceylon, the dry season may be the malarious one. Mosquitoes may breed in partially dried up streams, which in places become only a series of pools; with the advent of the rains the larvæ are washed away. A drought which causes famine may give rise to such conditions over wide areas. The Ceylon malaria epidemic of 1935 occurred in a season of drought which caused both famine and conditions highly propitious for excessive mosquito breeding.

Christophers (1911) in his studies of malaria in the Punjab, observed that in epidemics the heaviest mortality occurred among the poorest classes. He also noted that the spleen rate after an epidemic was highest among the poor. Of 12 great epidemics of malaria which devastated the Punjab in the last half of the nineteenth century and the early years of the twentieth, 7 followed seasons of famine or acute scarcity. Taking the price of food-stuffs as an index of scarcity, he found a high correlation between scarcity and mortality from fever; the epidemics of 1870, 1872, 1878, 1879, 1881, 1887, 1890, 1892, 1900 and 1908 all occurred during periods of high prices.

At the same time, Christophers demonstrated an equally high correlation between fever and rainfall and declared that "even if scarcity is in reality involved in epidemic causation, we should not expect to find it acting in the absence of the necessary factor of rainfall. We must not look for the effect of famine in this respect in the famine districts at the time of the famine, for at this time the essential factor, excess of rainfall, is absent." In the Punjab, years of scarcity or famine are usually followed by excessive rainfall and periods of high prices. The mere association of malnutrition and a high malaria mortality in such circumstances cannot be taken as proof that malnutrition *per se* is an important or essential factor in the causation of malaria epidemics. The relatively higher spleen rate in the poor can be explained by the fact that in India the poorest families often live in the outskirts of towns and villages and closer to mosquito breeding places. Poor children have scantier clothing than the children of the well-to-do and for this reason are more exposed to mosquito bites. The occurrence of malaria epidemics associated with "tropical aggregation of labour" was formerly attributed, in part at least, to the economic stress and privation suffered by poorly paid labourers. It seems more probable that the primary cause of such epidemics is that labour forces are exposed to infection with strains of malaria parasites to which they have previously had no opportunity of acquiring immunity.

Gill (1935) with experience of malaria epidemics in the Punjab and Ceylon has strongly emphasised the importance of "economic stress" as a factor in the causation of endemic and epidemic malaria. "In the case of communities in which hyper-endemic malaria is associated with economic stress, treatment by quinine or by other drugs is usually of little avail, and

the same remark applies with reservations to anti-mosquito measures. In such cases, it is clear that, whatever other measures may be employed in economically depressed areas, they must be accompanied, if they are to be effective, by measures designed to promote rural betterment by increasing the food supply and by relieving economic stress." (Gill, 1935).

In considering the relation between malaria and malnutrition there is another point of importance. Sinton (1935) and others have pointed out that malaria may itself be the direct cause of poverty and insufficient diet. A population heavily stricken with malaria is lethargic and cannot cultivate its land to the best advantage. A diminution in food supply and a fall in the standard of living results. One of the reasons why malaria and malnutrition are found in association is that the former leads to the latter.

The production of malaria in laboratory monkeys has enabled an attempt to be made to study the problem experimentally. In the Coonoor Laboratories two groups of monkeys, which had been for the previous two months fed, one on a first class mixed vegetarian diet with plenty of milk, and the other on a poor rice diet containing the minimum quantities of pulses, vegetables and supplementary foods were both subjected to experimental malarial infection. Although considerable variation was observed in the intensity of the malarial infections in individual animals, it was clearly demonstrated that primary infections did not differ significantly in the two groups, one well-nourished and the other in a state of malnutrition.

This experiment concerned only one aspect of the problem of the relation between malaria and nutrition. It confirmed general experience that the severity of first attacks of malaria in individuals and groups is not mitigated by a good state of nutrition. But how far the reaction of a community to malarial infection, as shown by the tendency to relapses, the spleen rate and the mortality rate from fever is influenced by the dietary factor remains a subject for speculation. The amount of anæmia occurring in a malaria-ridden population is unquestionably influenced by diet. The general problem is one on which no satisfactory pronouncement can as yet be made. Further research is required.

This discussion on the relationship between malnutrition and malaria has been long and must be left incomplete. However it has served to point out that the leading malariologists in India have demonstrated that an insufficiency of food and repeated attacks of malaria are both so interwoven into the social and economic problems of the people, that it is impossible to separate out and discuss each separately. The practical outcome of this is simple, but important. We cannot afford to attack the one without also attempting to deal with the other. This applies equally to a public health administrator responsible for the well-being of a large section of the population and to a physician

charged with the care of individual sick persons. Each will be wasting time and effort, if they attempt to eliminate malaria and neglect the state of nutrition or *vice versa*. Only a combined operation against the two can succeed.

DYSENTERY AND DIARRHOEA

Deaths from "dysentery and diarrhoea" figure prominently in the mortality statistics of India. In any hospital there are invariably numerous debilitated patients suffering from conditions, often of obscure origin, falling under this head. There is considerable evidence that "dysentery and diarrhoea", as widespread and fatal diseases, are closely associated with malnutrition.

The death rates ascribed to this cause in the Punjab, Orissa, the Central Provinces and the Madras Presidency (1936) are shown in Table 3.

TABLE 3

Deaths from "dysentery and diarrhoea" in various Provinces (1936).

Province.	No of Deaths.	Death rate per thousand.
Punjab	11,777	0·4
Orissa	16,383	2·3
Central Provinces ...	42,324	2·6
Madras	84,313	1·8

The death rate in the Punjab was from one-fourth to one-sixth that recorded in the other 3 provinces. Figures for 1936 are typical of other years. The staple cereal of the Punjab is wheat, milk intake is relatively large and standards of living are higher than elsewhere in India. There is less malnutrition in this province than in Madras, Orissa and the C. P. The two former are rice-eating provinces and surveys in various areas have shown that malnutrition is very common. In the Central Provinces, in which rice, wheat and millet are staples of about equal importance, poverty is extreme and the death rate the highest in India.

Throughout India the urban death rate from "dysentery and diarrhoea" is considerably higher than the rural. This suggests that grossly insanitary housing conditions are an etiological factor of importance. Malnutrition is particularly severe among the poorer classes in urban areas.

In times of famine there is invariably an increase in "bowel complaints", and the term "famine diarrhoea" is frequently encountered in reports. In the Report of the Famine Commission of 1901 monthly death rates per thousand from "dysentery and diarrhoea" in Berar in the 1899-1900 famine are given as follows:—

Month.		Death rate.	Month.		Death rate.
September	1892	0.8	May	1900	1.5
October	,,	0.7	June	,,	1.8
November	,,	0.7	July	,,	3.3
December	,,	0.8	August	,,	4.0
January	1900	1.0	September	1900	3.4
February	,,	1.0	October	,,	2.1
March	,,	1.3	November	,,	1.1
April	,,	1.2	December	,,	0.7

The famine was at its worst from June-September 1900. The death rate from "dysentery and diarrhœa" rose and fell in parallel with the severity of famine conditions.

Alexander Porter, Principal and Professor of Medicine, Medical College, Madras, published in 1889 an interesting but long since forgotten book entitled "The Diseases of the Madras Famine of 1877-78." Porter was in charge of famine sheds at the Royapettah Hospital in Madras and during the famine carried out 459 autopsies on patients who had died in the sheds. He recorded that 347 or 75 per cent of these died of "alvine fluxes" as a terminal condition. Ulceration of the gut was observed in about two-thirds of these cases. We may translate "alvine fluxes" into "famine diarrhœa."

Porter gives some records of the weights of his post-mortem cases which illustrate their extreme degree of malnutrition at death. The normal weight of Tamil men, he states, is 110-120 lbs; if a number of dropsical cases was omitted, the average weight of the famine cases at death was 77 lbs. For women the corresponding figures given by him are 95-105 and 77 lbs. The majority, he states, "are wasted until they were barely two thirds of their normal weight and when emaciation has reached this degree life is held by a slender thread which the least untoward circumstance is sufficient to snap."

The fact that intestinal lesions leading to severe diarrhœa often terminate the lives of famine victims is of considerable importance. If famine causes such conditions, it is reasonable to suppose that severe under- and mal-nutrition from which a considerable percentage of the population suffers in normal times, will have the same effect. In recent experiments carried out in Coonoor, monkeys fed on a typical "poor rice diet" showed a much higher incidence of diarrhœa than monkeys fed on diets based on whole wheat and containing milk and vegetables in abundance. The number of attacks was three times greater in the former group and the majority of the animals in this group eventually died

after suffering from diarrhœa for varying periods. The wheat and milk diet bore some resemblance to the diet of the Punjab, where the mortality from dysentery is much lower than in Madras and other provinces.

A full account of the clinical features of nutritional diarrhœa will be given in a later chapter. Here the main concern is to show that malnutrition probably plays a great part in the causation of the quarter of a million deaths that occur on an average every year in India from "dysentery and diarrhœa." If this proposition is justified, the public health importance of nutrition must be considerably extended.

RESPIRATORY DISEASES

This large group of causes of death includes the pneumonias and pulmonary tuberculosis, but here again we are dealing with an ill-defined group. It is known that the greatest number of deaths occur in the cold weather between December and March. It is probable that broncho-pneumonia is responsible for most of these deaths and that the two extremes of life, the very young and the very old are most affected. It has long been known that resistance to pneumonia at these ages is greatly impaired by poor nutrition and no doubt the majority of these deaths, especially in infants, are attributable to under-nourishment.

There is little exact knowledge about the prevalence of tuberculosis in India. But the disease is certainly increasing, especially in the towns, where large numbers of susceptible persons are brought together, often under most unhygienic conditions. Tuberculosis is likely to assume ever increasing proportions as industrialisation proceeds, unless adequate attention is paid to the rules of hygiene by all industrialists.

These include the provision of an adequate diet for the workers, as well as satisfactory housing and good working conditions. It is widely believed that dietary deficiency lowers the resistance to pulmonary tuberculosis and there is a wealth of clinical evidence to support this view. So well-established is it that the death-rate from pulmonary tuberculosis has been used as a measure of the nutrition of the people in European countries during the war. A rising tuberculous death-rate may certainly be taken to mean a falling standard of nutrition. There is little question that in India the present serious situation as regards tuberculosis is associated with an underlying dietary deficiency. It is foolish to expect that the new sanatoria and clinics opened under recent tuberculosis schemes will *per se* achieve very much. Such institutions are valuable and necessary, but no headway against this disease can be made until standards of education, living and nutrition are improved. Nothing less than a general raising of the standards of life for the whole people can withstand the advances of tuberculosis.

LEPROSY

Although leprosy is not one of the recorded causes of death, it is an important disease, closely associated with nutrition and may well be considered here.

The usual estimate of the number of lepers in India is about 1 million, or roughly 4 per thousand of the population. In certain areas the incidence is as high as 15 or 20 per cent. More than two-thirds of cases are relatively mild, belonging to the neuromacular type. Susceptibility to the disease is highest in childhood.

The geographical distribution of leprosy is described as follows in the Public Health Commissioner's Report for 1935:

"Leprosy is most commonly met with in certain parts of Southern and Eastern India In certain of the Himalayan valleys also, leprosy is very common. In Central India, its incidence is lower and in the North-west lower still. These variations may have some association with dietary conditions; the virile races of the north-west ordinarily use a well-balanced diet and show less leprosy than the less virile races of the south and east whose diet is relatively poor. It has been noted that a dry climate, hot in summer and cold in winter, is associated in India with a low leprosy incidence, but such areas are inhabited mainly by the more virile and well-nourished races. . . . It is a disease frequently associated with bad social and hygienic conditions and poverty, but these conditions are not always present."

Leprosy is relatively rare in the Punjab. A recent survey of 20 out of 29 districts revealed only 2038 lepers of whom 1005, or approximately 50 per cent, were from the Kangra district. This finding is of interest because malnutrition and food deficiency disease are common in the Kangra valley, where the diet is based largely on rice and contains little milk and few vegetables. Elsewhere in the Punjab the chief staple is wheat, and milk is available in reasonable abundance.

The idea that leprosy is closely associated with malnutrition has gained ground in India within recent years. Pronouncements to this effect have been made by lay Ministers of Health and other officials. The main argument in support of this view is the geographical and social incidence of the disease. But little or no convincing evidence pointing in the same direction has been obtained in India or elsewhere.

A diet survey of a group of families occupied in weaving, living in a suburb of Madras, all including cases of leprosy, was carried out by the Coonoor Laboratories in 1939. Investigations by Dr. R. G. Cochrane, then Chief Medical Officer, Lady Willingdon Leprosy Sanatorium, Chingleput, had shown

that the incidence of leprosy in this suburb was very high. The average intake of various food-stuffs per person are shown in Table 4.

TABLE 4

Diet of families containing cases of leprosy, Madras, 1939.

			(ozs. daily)
Rice (parboiled milled)	...	...	14.2
Pulses	...	...	0.9
Leafy vegetables	...	...	0.6
Non-leafy vegetables	...	...	2.8
Vegetable oils	...	...	0.5
Milk and butter-milk	...	...	1.1
Meat, fish and eggs	...	...	1.5

While it was of interest to find so severe a degree of malnutrition in a group with a high leprosy incidence, no very striking conclusion can be drawn. There would be no difficulty in finding plenty of groups in India, relatively free from leprosy, with as miserable a diet. On the other hand, it would be difficult to find a well-nourished group with a 5 per cent incidence of leprosy. The truth probably is that malnutrition either renders the body more susceptible to infection by the lepra bacillus, or facilitates the development of the disease after infection has taken place. It may thus encourage the spread of leprosy among children who are in contact with open cases. In other words, the part played by malnutrition in the etiology of leprosy may be analogous to the part it plays in the etiology of tuberculosis.

MORTALITY IN INFANTS AND CHILDREN

Infantile mortality rates are shown in Table 5, which includes also figures for England and Wales. A child up to the age of one year is called an infant.

TABLE 5

*Infantile mortality rates for British India and England and Wales, 1912-1936.
(Deaths per 1000 live births.)*

Period.		India.	England and Wales.
1912-1916	...	204	102
1917-1921	...	217	89
1922-1926	...	181	73
1927-1931	..	176	67
1932-1936	...	173	61
1937	...	162	58

(Public Health Commissioner's Report for 1936 and 1937.)

While the infantile mortality rates for recent years show some improvement on those of earlier decades, statistical examination of the figures for the decennium 1926-1935 failed to reveal a statistically significant reduction. The fact, however, that the recorded rate has not risen in spite of improved registration should be borne in mind.

In most provinces the urban rate is higher than the rural; for British India as a whole in 1936, the latter was 75% of the former. The difference is particularly striking in the Madras Presidency, in which rural and urban rates for 1936 were 152 and 281 per mille respectively. Registration in the Madras Presidency is more satisfactory than in many other Provinces. The rates (1936) for the 3 largest cities in India, Calcutta, Bombay and Madras, were 241, 250 and 218 per thousand live births respectively. The statement that about 1.75 million infants die annually before reaching the age of 1 year conveys a more vivid picture of the actual state of affairs than a table of mortality rates. About 50% of total mortality takes place during the first month of life, and of this about 60% occurs in the first week. Deaths in the first months are usually described in returns as "infantile debility, prematurity." Respiratory diseases account for a large number in the later stages of infancy. It is at present impossible to say, what percentage of the one and three-quarter million infants, who every year in India pass out of the world so soon after their entry, is attributable to faulty feeding. But there can be no doubt that it is a large proportion.

MATERNAL MORTALITY

That the maternal mortality rate in India is high is well-known to all those concerned with the care of women during pregnancy and childbirth. In the greater part of the country, however, accurate figures do not exist. In Madras, urban and rural rates for 1936 were recorded as 7.5 and 12.0 per thousand respectively, but such figures may represent an understatement. Throughout the country the female death rate during the reproductive period is higher than the male and the excess is largely due to mortality from child bearing.

In 1936 a special enquiry into maternal mortality was carried out in Calcutta by Neal-Edwards (1938). For the year ending June 1937 the remarkable high mortality of 24.5 deaths per 1000 registered live births was recorded. This figure is probably excessive for a number of reasons, such as the defective registration of births, the inclusion of imported cases and errors in assigning the cause of death. With all allowances made, however, it is clear that the rate is very high.

Causes of maternal deaths (Calcutta 1936-1937).

Cause of death.	Number.	Per cent.
Puerperal sepsis	224	31·95
Albuminuria and convulsions	126	17·97
Anaemia	165	23·33
All other causes	186	27·75

The considerable mortality from anæmia may be noted, since there is a close association between the anæmias of pregnancy and dietary deficiency.

No very definite conclusions can be reached about the relation between maternal mortality in general in India and malnutrition, but they are certainly closely related.

CONCLUSION

Some of the figures given in this chapter may appear dull and uninteresting at first sight. A table of statistics of morbidity lacks the emotional appeal of a sick person: yet it represents the sum of suffering of many persons and as such merits attention. A doctor in India must be familiar with the high death rate from fever and malaria, dysentery and diarrhoea, tuberculosis and the great losses in infancy, early childhood and the child-bearing period. They are such as one would expect in an ill-nourished people, ignorant of the rules of health. The melancholy state of affairs outlined in this chapter, with widespread manifestations of ill-health closely associated with malnutrition, must not be a cause for despair for the public good: still less can it be an excuse for individual lethargy. Rather it should be a stimulus to new and wider activities. There is every reason to suppose that an energetic application of scientific principles and methods to the traditional Indian way of life could provide the means to supply each individual with a sufficiency of food and a sound basis for a healthy life. Such an improvement can only be brought about by revolutionary changes in Indian agriculture, in the social and political life of the country and what must be faced, the religious customs and beliefs of many of the people. It is not the purpose of this book to discuss the necessary changes or to produce another "plan" for India. But any comprehensive state plan must have the backing and support of sound medical opinion. The role of the medical profession and its importance in public life is rapidly increasing. In addition to the ancient task of caring for the sick, we now have the additional duties of advising the people and the government on widespread social problems involving health. Every doctor practising in India must be aware of the conditions described in this chapter. His influence both in private and in public life must be exerted to help bring about such changes in the manner of life of the people, that optimum living conditions for health are available to all.

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CHAPTER 2

MALNUTRITION

A GENERAL DESCRIPTION

Definition. Malnutrition is a condition of ill-health, caused by a failure of the tissues of the body to obtain a sufficiency of the chemical substances essential for their normal metabolism and full activity. Malnutrition may be primary, in which case the food consumed contains insufficient quantities of the essential chemical factors, or it may be secondary, in which case the body, owing to some disease of an infectious or degenerative nature, is unable to digest or utilise in a proper manner the food which has been consumed. The principle features of malnutrition are a loss of sense of well-being, a general lack of energy and initiative, a failure in all-round efficiency, an inability to grow normally in the young and a loss of weight in the adult, which may in severe cases lead to a progressive emaciation. Malnutrition may be associated with certain definite physical signs due, in part at least, to deficiencies of specific factors; such signs include xerophthalmia, hyperkeratosis follicularis, angular stomatitis, nutritional glossitis and nutritional diarrhoea. The state of malnutrition includes varying grades from a slight departure from normal well-being and efficiency to a severe or even fatal disease with gross wasting and a general failure of the physiological functions of the essential organs.

INTRODUCTION

It will be observed that in the definition of malnutrition no attempt has been made to distinguish between malnutrition due to lack of specific dietary factors and that due to a general insufficiency of food (*e.g.*, partial starvation). In the literature of medicine there are many descriptions of the signs of disease purporting to be due to deficiencies in single essential food factors. Such cases do sometimes occur. A detailed description of the signs of deficiency diseases, following a classification based on a lack of separate individual dietary factors, may please the systematist and appear neat and concise. In practice in India, it is unusual for a single sign or symptom of malnutrition to be found alone. Inadequate food intakes of essential vitamins and minerals are in the majority of cases associated together with an insufficiency of total calories. In the resulting clinical picture, the features of a single deficiency may predominate, owing to individual circumstances, but a more careful examination will usually reveal the effect of a general deficiency of the diet. It is essential to stress the uniformity rather than the diversity of the symptom complexes and to emphasise that broadly speaking deficiency diseases fall within a single pattern, because in the important field of prevention and treatment, attention must be paid primarily

to both the quality and quantity of the food as a whole, rather than to deficiencies of specific factors. The use of vitamins and minerals in their pure state can only be an accessory form of treatment and can never be a substitute for a wholesome diet. To quote Sir John Megaw, "We must be on our guard lest vitamins should become a craze."

Standard text-books of medicine and tropical medicine for the last two generations have contained a section on Deficiency Diseases usually confined to accounts of beriberi, pellagra, scurvy, rickets and osteomalacia; a student of nutrition in India can derive only limited assistance from such a section. For he will soon observe that although direct and indirect evidence of ill-health due to an insufficiency of food is widespread amongst the people of all parts of the country, yet the diseases named above have only a limited distribution. Except in the northern districts of the Madras Presidency, beriberi is a rarity. Pellagra is uncommon except in occasional outbreaks amongst the small maize-eating section of the population. Scurvy is excessively rare, being usually only seen under famine conditions. Rickets and osteomalacia are largely confined to the purdah population in North Indian cities. In fact it is possible to have a wide experience of medicine and nutrition in India and to have seen none of these diseases. However, even these well-defined clinical entities rarely exist in "pure" forms as simple deficiencies of single factors. Many of the clinical features frequently found in patients suffering from these diseases can only be explained by an all-round insufficiency of food. For convenience of description these diseases will be considered separately in the next chapter. Here an attempt will be made to give an concise clinical description of the common signs and symptoms of malnutrition, as they present in large numbers amongst the population of all parts of India. The order of description is based on the manifestations as they occur in the different systems of the body. This is a more practical approach than a classification based on the effects of lack of separate individual essential food factors, which may be too academic for the practising physician.

ETIOLOGY

A breakdown in nutrition can theoretically arise either from a defective intake of essential nutriments, an inability of the intestines to absorb them or a failure of the tissues to utilise them. In practice all these possibilities occur, sometimes in the same individual. Occasionally a patient with a long history and well-developed signs of malnutrition dies and at post-mortem no evidence of any infectious or other disease is found. In such a case death is attributable to a failure to obtain the essential nutriments in the diet over a prolonged period of time. Alternatively, all the signs of malnutrition may be fully developed in a patient suffering from a chronic infectious disease, despite a satisfactory dietary intake. The physical signs of malnutrition can be fully and completely demonstrated in many cases of generalised tuberculosis, despite an adequate dietary history and with a satisfactory intake of protective foods.

No assessment of nutrition either in a single patient or in a large group of persons is complete without a good dietetic history and an estimation of the nature and strength of the parasitic infections present.

In general it may be said that the principal causes of malnutrition in India are a deficient total food intake (inadequate calories), chronic malaria, a deficient consumption of milk and vegetables (inadequate amounts of vitamins A and the B₂ group) and chronic intestinal infection with protozoal and helminth parasites. There are, of course, numerous other factors which may be operative.

GENERAL FEATURES

Two of the earliest and most prominent features of malnutrition are a loss of energy and a loss of weight. Listlessness, lack of enterprise and disinterestedness are important evidence of early malnutrition. An observer may readily detect these amongst groups of school children, labourers or soldiers. In the very young, a sign of inadequate nutrition is a failure to get into mischief. The listless attitudes and quiet demeanour of children, sitting patiently and uncomplaining around relief works and camps in famine areas without any spontaneous games or play, are characteristic manifestations of their poor nutrition. A few minutes' observation of the energy and initiative displayed by a group of labourers at work or a party of soldiers carrying out fatigues can provide valuable evidence for assessing their nutritional state. Severe cases of malnutrition in hospital often display a complete lack of interest in themselves and their surroundings. There may be insufficient initiative to attempt to eat and a total absence of any desire to get better.

Loss of weight in an adult or a failure to gain weight in a child is a constant and characteristic early sign of malnutrition. The weighing machine is an invaluable guide to a schoolmaster in assuring himself that his children are receiving adequate amounts of food. It is equally useful to officers in charge of recruits-training depots or doctors responsible for an infant or children's clinics. It is generally possible to get a good idea of the nutrition of a patient seen for the first time by picking up a fold of skin on the abdominal wall or over the pectoral muscles. The quantity of the subcutaneous fat, the laxity of the subcutaneous tissues and the underlying muscle tone can provide an indication as to whether there has been any recent loss of weight. In severe malnutrition as much as forty or fifty pounds may be lost: there is often an almost complete absence of subcutaneous fat and great wasting of voluntary muscle. Emaciation may be so marked that patients can be aptly described as living skeletons.

Dehydration is frequently associated with malnutrition. In many instances this is no doubt due to an inadequate water supply and difficulties involved in getting it. But this is not always so. The starving destitutes that

roamed Calcutta during 1943 were frequently severely dehydrated. Yet Calcutta has an abundant water supply, opportunities for drinking being available at almost every street corner. Patients with malnutrition in hospitals readily become dehydrated unless steps are taken to ensure an adequate fluid intake. The normal stimulus of thirst appears to be in obeisance. Œdema, presumably of nutritional origin, associated with low plasma proteins is seen not infrequently. It may be generalised, but is usually more extensive in the lower limbs. In severe cases the whole mechanism of the body for the regulation of water metabolism becomes disordered. On more than one occasion dehydration and œdema have been seen co-existent in different parts of the same individual. For this reason intravenous hydrotherapy, which is frequently necessary, must be carefully watched.

An apology is perhaps needed here for expounding the obvious. It may be argued that every one knows that an ill-nourished person loses weight, becomes thin, is easily tired and devoid of energy and initiative. However, recent medical literature contains many descriptions of complex chemical and physiological tests, involving elaborate apparatus for assessing nutrition and has emphasised the difficulties and errors that may be met. As a result many doctors, ignorant of the principles involved in such tests and without the means of carrying them out, have lost confidence in themselves and fail to use the simple observational methods. They become afraid or unwilling to express an opinion in nutrition and are all too ready to summon the specialist. In India at least, this is unfortunate. Practical nutritional assessment seldom requires laboratory facilities or expert knowledge. It has been found that a competent opinion on nutritional status can in almost every instance be given as a result of simple clinical examination, in which the above elementary and commonsense observations play a large part.

THE SKIN

Hyperkeratosis (Nutritional). An early and frequent manifestation of defective nutrition of the skin is a proliferation of the cells of the epidermis giving rise to a superficial hyperkeratosis. There is usually some degree of atrophy of the sebaceous and sweat glands. As a result the skin becomes dry, rough and scaly. In slight cases only a small departure from the smooth, moist, velvety texture of normal skin may be apparent. Grades of increasing severity are found up to a very advanced condition, described as crazy-pavement skin by observers in Africa (Trowell, 1940). This condition is not uncommon in India and the name provides an excellent description of its appearance. Irregular cracks occur all over the dry hard epidermis. Most of these are superficial and are usually 2 or 3 mm. apart, but quite irregular in pattern. Deeper cracks sometimes occur at wider intervals and these may be infected and ulcerated. The superficial dead layers of the squamous epithelium flake easily. There are usually small irregular areas of hypo- and hyper-pigmentation completely breaking up the normal uniformity of the skin

and adding to the "crazy" appearance. All degrees of skin signs are more marked on the extensor surfaces of the limbs, but they may become universal. The face is usually least affected. Irregularity of pigment production is common. Symmetrical areas of increased pigmentation of the face are not infrequently seen. Hypo-pigmentation occasionally occurs. Unlike pellagra the lesions are not photo-sensitive. Itching is characteristically absent. These very severe forms of crazy pavement skin are only found in association with other manifestations of malnutrition. Secondary infection both mycotic and bacterial is common. Scabies is very frequent in cases of malnutrition. It was one of the principal clinical features of the Bengal Famine.

Follicular Hyperkeratosis. A distinct clinical type of hyperkeratosis occurs when the hair follicles are affected. The mouths of the follicles become filled with plugs of desquamated keratin, which project above the surface of the skin in the form of small papules about 1 mm. in diameter: the papules are firm in consistency and do not pustulate. This condition, which is known as follicular hyperkeratosis, gives rise to a characteristic appearance and sensation to touch. In a well developed case, on passing the finger tips along the outer surface of a child's arm, there is a characteristic sensation of roughness, as the surface of a grater. The numerous little papules have been considered to resemble the skin of the toad and the condition has accordingly been called phrynoderma. Again the extensor surfaces of the limbs are most affected, but the whole trunk may be involved. The face, genito-anal region, hands and feet are usually spared. The condition is much more frequently seen in children than in adults. It is not common in North India, but in certain districts in South India it is very prevalent. The condition is definitely associated with a poor diet lacking in fresh vegetables and milk. It is also frequently associated with xerophthalmia and other signs of malnutrition and it is very rarely found in children of the well-to-do, living on a well-balanced diet. Spectacular improvements have often followed vitamin A therapy, but occasionally no response is observed. There is no doubt that for practical purposes in India follicular hyperkeratosis should be considered as a deficiency disease and treated accordingly. The exact conditions, dietary and otherwise, under which it arises are however not yet fully understood and it has been reported in English children, whose food intake was in everyway satisfactory.

Muco-cutaneous Degeneration. A cutaneous manifestation of malnutrition is a peculiar degeneration that affects the junction of the skin and mucous membranes. All such junctions may be affected. The lips, the nostrils, the eyelids, the external auditory meatus, the prepuce of the penis, the vagina and the anus may all be involved. The lesion has essentially the same features at all sites. The mucous membrane over the junction becomes white, thickened and sodden and cracks appear. The white patches may extend inwards into the oral cavity, over the surface of the glans penis or into

the vagina, where a leucorrhœa may be set up. The cracks may be the seat of chronic infection and are then painful. The angle of the mouth is the site of selection, which is always affected first and an associated glossitis is usually present: hence the condition has been described as angular stomatitis. Cracking of the lips is a frequent and early feature and is known as *cheilosis*. The genital lesions are rarely, if ever, present alone and generally only occur in advanced cases. In rapid nutritional surveys in schools there is no necessity to inspect the genitalia, since the lesion if present will always be manifest round the mouth. Angular stomatitis is the commonest of deficiency diseases. Amongst a rice eating population it breaks out with remarkable regularity whenever the supply of fruits, vegetables, milk and meat fails. It disappears as soon as the intake of these protective foods rises. It can readily be cured by skimmed milk and yeast and hence is attributable to the lack of B₂ group of vitamins. Riboflavin often brings about marked improvement and the condition is undoubtedly closely associated with this vitamin, but probably other factors in the B₂ group play a subsidiary part.

Eczema of the Genitalia (Nutritional). In both sexes a severe eczema involving either the scrotum and penis or the labia majora, labia minora and clitoris may arise from nutritional defects. The skin over these regions becomes thickened, rough and stiff and is covered with dry scales. Occasionally the surface may break and a raw, red, weeping eczematous area be formed. In contrast to other nutritional lesions of the skin, it is intensely itchy. Scratching may lead to secondary infection. The area involved always has a well-defined edge and is sharply limited to the genitalia. The condition is usually associated with angular stomatitis and other signs of muco-cutaneous degeneration, especially lesions of the prepuce and vagina. It usually occurs in persons living on diets grossly deficient in protective foods and very poor in the B₂ group of vitamins. It can usually be relieved by yeast therapy or an all-round dietary improvement.

THE EYES

Xerophthalmia. This condition, also known as keratomalacia, is probably the most frequent sign of malnutrition which is seen in India. The description given by Wright (1934) cannot be improved upon: "The earliest manifestation of keratomalacia is night-blindness. The next evidence of this progressive deficiency is a peculiar smokiness of the conjunctiva; this is the earliest obvious sign just as the night-blindness is the earliest symptom. One may meet either or both of these in a youth who is apparently healthy looking in other respects. They are, however, danger signals. When the early signs and symptoms appear, if nothing is done to augment the amount of essential food factors in the diet, the case rapidly progresses and marasmus sets in. *Pari passu* with the wasting there is an advance in the eye symptoms. As a rule, the smoky conjunctiva becomes dry and wrinkled, greasy looking, the cornea becomes dull and lustreless and eventually opaque. Later the cornea

undergoes necrosis and ulceration and, if untreated, the ulcer perforates and the eye is ultimately lost." The condition is frequently associated with Bitot's spots. These are glistening white plaques of dead conjunctival epithelium usually triangular in shape and firmly adherent to the underlying conjunctiva. They are generally bilateral and are surrounded by a dense brown pigmentation.

Early xerophthalmia is common in the rice eating areas of India: in parts of the Madras Presidency as many as thirty per cent of the children have been found affected. Although in the great majority of children the condition is arrested before serious defects of vision arise, yet in many cases the disease progresses. All authorities are agreed that many thousands of persons in India are permanently blind from this cause. Wright from his experience at the Government Ophthalmic Hospital, Madras, considers it the most important cause of blindness in childhood and youth.

The early signs of xerophthalmia, dryness, increased wrinkling and pigmentation of the conjunctiva and the associated night-blindness are undoubtedly closely related to dietary defects, especially an inadequate intake of vitamin A, in children. In adults the interpretation is more difficult. They are not infrequently seen in persons, who are living on adequate diets, are unassociated with any visual defects and are not necessarily progressive lesions. In some cases they may be residual scars of an old lesion, which has been inactive for many years. In others it would appear possible that repeated attacks of conjunctivitis may be responsible. War experience has shown that, although vitamin A deficiency undoubtedly leads to night-blindness, by no means all night-blindness is due to dietary causes and many other factors, including psychological strain, may be responsible. Nutritional night-blindness responds promptly to vitamin A therapy.

Nutritional Amblyopia and Superficial Keratitis. In recent years two other eye manifestations of malnutrition have been reported. The first is a defect in vision amounting in some cases to total blindness which may or may not be associated with pallor of the optic discs and optic atrophy and other nervous degenerations. This nutritional amblyopia usually follows upon long periods on deficient diets (many of the cases described have been prisoners) and is often associated with other signs of malnutrition. There is a steady improvement in vision in most cases when the patient is put on a good diet, and both vitamin A and vitamin B concentrates have been reported to accelerate recovery.

The second is a superficial keratitis: the patients complain of burning sensations in the eyes, mistiness of vision and photophobia. Various degrees of circumcorneal injection and corneal vascularisation can be seen in most

A slit-lamp is necessary for detecting mild degrees of corneal vascularisation. The differential diagnosis from simple congestion or even interstitial keratitis is difficult for the inexperienced. The condition is almost invariably associated with cutaneous and alimentary signs of malnutrition and sometimes responds promptly to riboflavin therapy. In the writer's experience these two conditions are not commonly associated with severe malnutrition, as observed in general hospitals in India, nor do they appear common in North India or Bengal. That they are not infrequent in the South is shown by the records of the Government Ophthalmic Hospital, Madras, where both have been extensively studied (Aykroyd and Verma 1942; Verma 1942).

THE DIGESTIVE SYSTEM

Nutritional Glossitis. The tongue is swollen and enlarged, occasionally to such an extent that it is continuously pressed against the lower jaw and well marked dental impressions are visible. The organ is characteristically red. The mucous membrane may be seen to be desquamating in patches leaving a red raw surface visible. The papillæ are often unusually prominent.

Deep irregular fissuring is a common feature. Occasionally the tongue is seen to be small with a smooth atrophic mucous membrane with fine fissuring. Irregular patches of dark pigmentation are frequently present. The tongue is often extremely painful. So much so that fear of pain may prevent the patient from eating. This condition is closely associated with angular stomatitis and like it frequently arises, when inadequate supplies of milk, fresh vegetables and other protective foods are available. It is ascribable to a lack of the B₂ group of vitamins and especially nicotinic acid.

Nutritional Diarrhœa. Diarrhœa is a most frequent and important symptom of malnutrition in India, often paving the way for much chronic ill-health and sometimes for a fatal illness. It is rarely so acute as in the bacillary dysenteries. Six to eight times a day is the usual number of motions. The diarrhœa is always worse in the morning and the bowels may give no trouble after mid-day. Sometimes the intake of even small quantities of food sets off an evacuation. The stools are usually watery, fæcal in colour and often contain visible particles of food, completely undigested. Microscopically undigested starch grains, vegetable fibres etc., can be readily seen. Inflammatory exudates are characteristically absent. Excess fat is not often visible. The large, pale, fatty, offensive stool described as characteristic of sprue in Europeans is only rarely seen in Indians. The nature of the diarrhœa is such that it would appear to be the result of a "small-intestinal" hurry rather than any inflammatory process. Although the diarrhœa can usually be controlled temporarily by sulphonamides, especially sulphaguanidine, and sometimes by nicotinic acid, relapses are frequent. It is usually the most striking and important feature of severe malnutrition, associated with great wasting, "crazy

pavement" skin and the appearances of a living skeleton. It is generally the principal cause of death.

At post-mortem a general atrophy of the whole intestine is usually seen extending throughout from the mouth to the rectum. All coats, mucosa, sub-mucosa and muscle are involved. The small intestine is the most severely affected, becoming progressively thinner from the duodenum down to the ileo-cæcal junction. Indeed the last two feet may be so thin and transparent as to resemble the texture of tissue paper. The mucous membrane of the small intestine is smooth and atrophic and clearly its digestive and absorptive functions must be seriously impaired. Ulceration of any part of the gut is uncommon.

The intestines of sepoy, who had died with chronic diarrhoea and malnutrition after evacuation from the Burma battlefront, presented an identical post-mortem appearance with the intestines of monkeys, who had died in a similar emaciated condition in the Coonoor laboratories, following the consumption for many weeks of a diet consisting of rice with minimal amounts of protective foods. The same gross atrophy of the small intestine was present in the experimental animals. A detailed pathological account with a full histological report of these monkeys has been given by Radhakrishna Rao (1942). The following is an extract of his report. "Degenerative changes were constantly present in Auerbach's plexus. In most cases the plexus was enlarged and œdematous, and hence easily recognised in the sections. Varying degrees of degenerative changes from cloudy swelling to complete degeneration, were found in the ganglion cells. The number of ganglion cells involved in the degenerative process and their distribution in the plexus showed great variation in the several specimens. In chronic cases, normal ganglion cells were rarely seen and the plexus was represented by empty spaces containing skeletons of dead and degenerating ganglion cells, round cells, fibroblasts and glial cells." The opportunities available for histological study of human material have been limited, but these findings have been confirmed in every case examined. All modern physiological research emphasises the importance of the autonomic nervous system in controlling the functions of the alimentary canal. An early degeneration of the neurons in this system might well be responsible for a complete breakdown of the digestive processes, severely limiting the absorption of nutriment and leading to malignant malnutrition, refractory to treatment. Such a degeneration may well be an initial pathological event in a vicious circle of defective food take, intestinal atrophy, defective absorption, further atrophy leading to the tissue paper small intestine and progressive emaciation.

THE CARDIOVASCULAR SYSTEM

In minor degrees of malnutrition there are no clear cut signs of cardiovascular disease. In more advanced cases there is a characteristic fall in blood pressure, which may be very low. A systolic pressure of 70 mm. and a diastolic

pressure which is unreadable are sometimes found. These low levels are always associated with other evidence of severe malnutrition and carry a poor prognosis. Cardiac failure with œdema is sometimes a terminal event. Ordinary cardiac stimulants are valueless and cases often end fatally after dragging on for many weeks. At post-mortem the whole heart is found to be very small with marked atrophy of the muscle.

The hypotension, emaciation and pigmentation may present a picture strikingly resembling Addison's disease. However, no improvement has followed in a few cases to whom cortical extracts were given. The adrenal glands are usually small and atrophic, but present no specific changes.

THE NERVOUS SYSTEM

The principle nervous manifestation of malnutrition is a peripheral neuritis. This is indistinguishable from beriberi and will be considered in detail with that disease in the next chapter. Outside the endemic areas of beriberi, the condition is not common. It usually occurs in association with severe diarrhoea and wasting and is seldom the primary complaint.

Another interesting nervous condition is the "Burning Feet" syndrome, which has recently been investigated by Dr. C. Gopalan in Madras. It is not uncommon in other parts of India. The patients usually show obvious evidence of malnutrition, many of them suffering from angular stomatitis and eczema of the genitalia. Gopalan's description of these cases is as follows:

"A burning sensation in the extremities is the characteristic feature of these cases, and it is to obtain relief from this that the patient comes to the hospital. The burning sensation is usually confined to the soles of the feet, but is present in some cases, in the palms of the hands as well, patients describe their complaint by the Tamil words 'Kal erichal' which literally mean burning of the feet. No cases in which the hands alone were the sites of burning sensation have been met with. Characteristically, the sensation of burning starts in the soles of the feet round about the ball of the great toe and seems to spread therefrom to involve the soles completely. In some cases, the burning sensation may spread upwards to involve the dorsum of the feet and the ankle region. The burning may be quite mild and tolerable, but is frequently most intense and incapacitating. It may come on in paroxysms, the patient having excruciating agony for a short period and then remaining free from the symptom for the rest of the day. Some patients seem to obtain relief by wrapping their feet in a wet cloth. Others find the contact of their feet with cold or hot objects intolerable and prefer to leave their feet alone."

In contrast to the marked subjective symptoms, physical signs of disease of the nervous system are rarely found. There is no loss of sensation or muscular power. In certain cases the affected areas suffer from a severe hyperidrosis, visible beads of perspiration standing out and the feet being moist to touch.

The condition can be relieved in 3 to 4 weeks by yeast therapy, but not by pure vitamin B₁, nicotinic acid or riboflavin.

A nutritional amblyopia has already been described. In returned prisoners of war, neurological signs referable to spinal cord lesions have been found in association with amblyopia. Fifty-six such cases of spinal ataxia were found amongst Indians, who had all been prisoners in Japanese hands for at least two years. There can be little doubt that these were nutritional in origin. No account of this condition in civil practice has been seen and it is probably rare. Sub-acute combined degeneration of the cord is uncommon in Indians.

The severe depression often associated with malnutrition in India has already been described. It is the main psychological feature. Manic symptoms and dementia, important accompaniments of pellagra, are rarely seen.

THE BLOOD AND URINE

Anæmia is an important feature of certain cases of malnutrition. The subject is dealt with fully in Chapter 4.

In severe cases of malnutrition, especially those associated with diarrhoea, there is frequently a fall in serum proteins. The normal value for plasma proteins is 7 gms. per 100 ml. and figures much below this may be found in malnutrition. Oedema usually occurs when the level falls between 3.5 and 4.0 gms.

There is an extensive literature dealing with the vitamin content of both blood and urine. Numerous attempts have been made to correlate nutritional status with changes in the concentration of different vitamins in the blood and urine. The chemical methods involved are for the most part outside the scope of the practitioner: the value of individual tests in the diagnosis and assessment of nutrition have, as yet, not been satisfactorily established. In India, at the present time, assessments of nutrition, satisfactory for all practical purposes can be made by physicians using clinical methods alone, without involving special biochemical and physiological tests.

Before closing the general description of malnutrition in India, reference must be made to two diagnostic terms, sprue and ariboflavinosis, both of which are widely used in medical literature to depict certain nutritional syndromes.

Sprue is a condition clearly very similar to the picture of malnutrition already described. It is a diagnosis not uncommonly applied in India to European and Anglo-Indian patients, but rarely to Indians. The features usually ascribed to sprue are those of a wasting disease, associated with glossitis, macrocytic anaemia and diarrhoea with the passage of pale, large, frothy offensive stools. Only in the last respect does it differ from the picture of malnutrition as usually seen in India. Otherwise it complies in detail with the general description of multiple deficiency diseases. The character of the stool is by no means an absolute racial distinction. The typical sprue stool is occasionally seen in an Indian and sometimes absent from an otherwise typical European case. The nature of the stool probably depends more on the quality of the diet and the type of intestinal flora than on the pathology of the disease. There can be no

question that sprue is a mixed deficiency disease and that the general clinical picture is the same as that of malnutrition in Indians. The difficult problem is the nature of its etiology and epidemiology. In Europeans the disease is only occasionally associated with a defective food intake and in some instances there is epidemiological evidence suggesting an infective agent. The introduction of the linguistically mixed term, "para-sprue," which is recently becoming popular as a label for diarrhoea of nutritional origin in India seems an unnecessary complication of nomenclature. Nutritional diarrhoea is a sound descriptive diagnosis, preferable in most instances to "sprue", a Dutch word apparently meaning an inflammation of the tongue.

The diagnosis of ariboflavinosis or hyporiboflavinosis, first introduced in America, is becoming widely used. Many signs such as angular stomatitis, cheilosis, generalised mucocutaneous degenerations, genital eczema and superficial keratitis have been included by different workers, based on therapeutic response to riboflavin. Experience in India has shown that all these conditions do sometimes respond dramatically to riboflavin, but that the effect is erratic and inconstant. Further complete cure is seldom obtained without a generalised dietary improvement and relapses are frequent. Riboflavin deficiency can only be a part of the etiology of these diseases. A pure riboflavin deficiency probably rarely arises under natural conditions. It would seem better to preserve the descriptive diagnoses rather than to introduce a new term involving an incomplete description of the etiology of the conditions.

TREATMENT

This section will begin with an account of the methods available for the treatment of patients who have been sufficiently ill to merit admission to hospital. References will be made at the end to the treatment in the field or in out-patient departments of the innumerable minor cases, who do not require hospitalisation or for whom hospital beds are not available. Such medical advice as can be given is, in practice, limited in these cases largely by economic factors.

General. Severe malnutrition is always associated with mental depression and retardation. Many patients appear at first to have no desire to get better or even to live. All treatment is of no avail, unless the co-operation of the patient is obtained and until he can be persuaded not only that it is a good thing to get better, but also that it is possible for him to be cured. To secure a right psychological attitude is the first essential of treatment. The patient's confidence must be won. He must be made to realise that the circumstances, which reduced him to his present condition, are now past. An energetic and persuasive Ward Sister can work wonders and is probably the most important single factor in recovery. Attractively served food, a bright and cheerful ward with simple decorations, a willing ward staff who are prepared to spend time and patience coaxing refractory patients to eat, these are the important elements of treatment and many Sisters have been able to achieve much along such lines with extremely limited material. A cheerful convalescent patient, who has himself recovered from severe malnutrition, may prove invaluable in encouraging and helping bad cases. An active diversional therapy department is a most valuable asset.

Dietetic. Elaborate and rigid dietary scales should be avoided. As far as possible each patient should be considered separately and his whims and fancies pandered to. They are often a better therapeutic guide than the physician's dietetic theories and should always be respected. It is important that feeds should be frequent and small. Some nourishment should be taken at least six times a day. It is generally convenient to have three main meals a day and to give milk drinks, suitable fruits, fruit drinks, sweets and sweetmeats in the intervals. At first the main meals should be small, especially if diarrhoea is a prominent feature. Overloading the stomach will excite an oversensitive gastro-colic reflex, cause flatulence and abdominal distress. Details of diets will not be given. These depend on the customs and taste of the people and the available food. In the past too much attention has probably been placed on the exact ratio of protein, fat and carbohydrate in the diet. The malnourished person usually has an aversion to fat and this should be limited. Otherwise it may be said that any wholesome well-cooked food is valuable in small quantities. As a guide for those, who may be responsible for organising hospital catering, the scale of hospital diets for Indian troops used by the army is given in appendix 4 and the menu of the Special Invalid Kitchen of a large army hospital in appendix 5. These both proved suitable and practical under the special army conditions. Local modifications to meet the customs of the people and the available supplies could readily be made. Pulses and coarse fruits are usually poorly tolerated and can often be seen passed completely undigested into the bed pan. Meat and flesh foods are valuable and every effort should be made to encourage patients to eat them. However, they are not essential and need not be pressed against strong religious conviction. Milk and milk preparations are usually the sheet anchor of treatment. Curds are particularly valuable and are relished by all Indian patients. Tinned milk makes excellent curds.

Patients, who are first seen in a badly collapsed state, should not be given solid foods. Many fatalities have occurred during famines through sudden overloading of the stomach. Such patients should receive only a fluid diet for 48 hours or until their condition improves. Small quantities of diluted, citrated milk or sweetened fruit juice should be given hourly. Later an egg beaten up in milk may be added. For very weak patients unable to feed themselves continuous intragastric drip feeding, which may be carried on for many hours is convenient and a great saving of labour for the nursing staff. Dehydration may have to be overcome by the intravenous infusion of salines to which glucose may be added. For the treatment of hypoproteinaemia, both concentrated solutions of serum proteins and protein hydrolysates have been used. Krishnan, Narayan and Sankaran (1944) gave over 3000 intravenous injections of a papain digest of meat to about 1000 cases of starving destitutes in Calcutta and believe that this was responsible for the saving of some lives, but the conditions of work made accurate assessment of its value impossible. Lowe (1946)

stated that, in the experience of the School of Tropical Medicine at Calcutta, the results of intravenous therapy with proteins and protein products were on the whole disappointing. The practical value of this form of treatment in India is clearly still *sub judice*. In the treatment of nutritional œdema associated with hypoproteinæmia, mercurial diuretics have been found to be of value by numerous physicians.

Vitamin Therapy. Vitamin A preparations are valuable especially in children. The various skin conditions previously described especially follicular hyperkeratosis are usually improved by vitamin A. Sometimes spectacular successes are rapidly obtained: more often improvement is slow and follows only upon several weeks of treatment. Night-blindness is often rapidly relieved. A single dose of 200,000 International Units will usually produce improvement in a few hours. The signs of keratomalacia improve more slowly. There is usually a general improvement in the health and well-being of the child. In adults the value of vitamin A therapy is much less.

Vitamin B₁ (thiamin) therapy is discussed in the section on beriberi in the next chapter.

Nicotinic acid will often give spectacular results in relieving the pain associated with glossitis and stomatitis. New mucous membrane is rapidly formed over the tongue and the pain present during chewing and swallowing diminished. Nutritional diarrhœa is also rapidly controlled in many cases by nicotinic acid. A dose of 100 mgm. three times a day by mouth is generally adequate, but some cases only respond when the dose is given by subcutaneous injection. If any benefit is going to occur, signs of improvement are usually manifest within 48 hours. In the absence of signs of improvement it is valueless to persist with the vitamin longer. Although dramatic relief of symptoms is frequently obtained, in other cases the stomatitis and diarrhœa appear completely unaffected by the vitamin. It would appear at present impossible to forecast by clinical examination, which cases are likely to improve and in which the symptoms are likely to remain unaffected. In my experience not more than fifty per cent of patients are benefited. Although rapid relief often occurs, complete cure is seldom effected by nicotinic acid alone, unless an all round improvement in the diet is simultaneously effected and relapse is frequent unless this is maintained. To sum up, it may be said that the nicotinic acid is a valuable, but uncertain therapeutic weapon.

Riboflavin has not been available in India hitherto for therapeutic purposes except on a small scale. It has been shown to be effective in relieving the signs and symptoms of superficial keratitis. Many cases of sore mouth with excoriations at the angles and with other manifestations of muco-cutaneous degenerations and genital eczema have responded promptly to riboflavin. A dose of 5 to 10 mgm. daily by mouth or subcutaneously is usually adequate.

In general the comments made on nicotinic acid therapy, apply also to riboflavin.

Although clinical evidence of scurvy is very rare in India, most cases of malnutrition have been living on diets containing barely minimum amounts of vitamin C. There is therefore every indication for providing liberal quantities of vitamin C. This can best be done by giving fruit or fruit juices rich in the vitamin. If these are not available, ascorbic acid (50-100 mg. daily) should be given. Sprouted grams and pulses are also a valuable source of the vitamin.

Yeast preparations such as marmite and dried yeasts have already been mentioned. They are most valuable for the treatment of minor manifestations, especially stomatitis, in out-patients. Undernourished children are frequently benefited. Results with severe cases of malnutrition in hospital are often disappointing. If an adequate supply of protective foods is available in the diet, supplementing the normal source with the concentrate in yeast appears to be of little value. Perhaps the patient is unable to absorb or utilise the extra vitamins. Yeast and its preparations are distasteful to sick Indians and it is now the writer's custom to prescribe them only when a hospital dietary is not completely satisfactory. In India it is probable that yeast therapy will have its maximum beneficial effect in out-patient clinics and field treatment.

In recent years vitamin therapy has been extensively used in the treatment of malnutrition. Pure vitamins have been given both parenterally and by mouth and many preparations of vitamin concentrates have been employed. Although vitamin therapy is valuable and has its place in the treatment of malnutrition, results are on the whole disappointing. In tropical practice the slow and uncertain improvement following the exhibition of pure chemicals to patients with signs of malnutrition contrast strongly with the prompt and spectacular results usually obtained from the use of the appropriate chemotherapeutic remedy in acute infections. Vitamin therapy can only be supplementary and can never, under any circumstance, satisfactorily replace a defective supply of food.

Treatment of Infections. It cannot be too strongly stressed that no dietetic treatment of the malnourished can be effective in the presence of infections. An essential part of the treatment of malnutrition consists in a diligent search for protozoal and helminth parasites and in their eradication. Experience has shown that protozoal diagnosis is often difficult in the undernourished. Although a patient search may have failed to find malarial parasites and amœbæ, in some cases prompt improvement follows anti-malarial and anti-amœbic treatment. The presence of persistent unexplained anæmia or diarrhœa is an indication for the use of these remedies, even in the absence of an exact diagnosis. The sulphonamides, especially sulphaguanidine, are generally effective in controlling nutritional diarrhœa, although the effect is

usually temporary and relapse frequent. In emaciated persons very small doses are often adequate.

Following severe malnutrition recovery is always slow. Even under the best hospital conditions there is always a definite mortality. In advanced cases it would appear that irreversible degenerative processes have taken place. Convalescence must never be hurried and after severe illness activities must be limited. Unexplained sudden deaths have occurred in patients who appeared fully convalescent and who were up and about in their wards. Some patients can never regain their full health and relapses are frequent, if the patients are again subjected to nutritional strains. Soldiers, who have been evacuated to base hospitals on account of nutritional breakdowns, should never be sent on active service again.

Surgical Patients. Experience in the recent war has again emphasised the importance of an adequate diet for the satisfactory repair of wounds, both those made by the surgeon and those resulting from accidents and enemy action. However expert the technical skill exhibited in the operating theatre, the beginnings made there by the surgeon to ensure efficient wound healing, with no unnecessary sepsis, can only be carried on in the wards, if the injured tissues are supplied with all the essential chemicals necessary for wound healing. Laboratory experiments have shown the importance of vitamin C and protein in wound repair. Good results will only be obtained in surgical wards if the patients receive well-balanced diets rich in these substances. There should be no need for extra vitamin therapy, if the food going to the wards is satisfactory in every respect, both in quantity and quality.

Out-patients. Large numbers of patients attending the various out-patient department of hospitals and the clinics of private practitioners suffer from minor manifestations of malnutrition, in addition to the complaints for which they are seeking medical advice. The insufficiency of their food may be an important contributory factor in their illnesses causing or prolonging their disabilities. Such patients present a difficult problem to their doctor. He may give them good advice on what to eat and recommend a more liberal consumption of milk, ghee, fresh vegetables, fruits and other foods, but such counsel is usually thin air and completely impractical in the patient's economic status. He can, however, often help to husband the patient's limited resources and suggest minor economies in their way of life, and can suggest that such money as is available might be spent on a few little extras, even luxuries, of food rather than in the chemist's shop. There are also two potent remedies, which can be made freely available in India and from which the majority of outpatients would derive more benefit than from many of our present day stock mixtures. These are fish liver oils and yeast preparations, which can be valuable extra sources of vitamin A and D and the B Group of vitamins in addition to those present in the food. This particularly applies to certain clinics. Dr. Gurubathan who

was for many years in charge of the large Swedish Mission Eye Hospital at Coimbatore observed empirically that the liberal distribution of cod liver oil was essential to the running of his out-patient department. A large proportion of ophthalmic diseases including trachoma, were found not to respond satisfactorily to accepted remedies, unless the patient received vitamin A therapy concurrently. There is little doubt of the general validity of these observations in many Indian eye clinics. A similar generalisation is probably applicable to skin departments.

In the present poor state of nutrition of so many of the out-patients attending hospitals all over India, but especially in the rice growing areas, fish liver oils and yeast can justifiably be freely given as tonics to a wide variety of patients in addition to those with direct evidence of malnutrition. Thus extra vitamin A and D can be prescribed for all children, who are underweight, for persons with abnormal dryness or roughness of the skin, for cases of chronic sepsis, in addition to cases of early xerophthalmia, follicular hyperkeratosis and obvious rickets. Extra vitamin B can help most pregnant and nursing mothers, children who fail to thrive and lack energy, cases of chronic low grade infections, patients with unexplained mild diarrhoea and glossitis, in addition to all cases of angular stomatitis and genital eczema.

A satisfactory daily dosage for such cases is half an ounce (approximately 15 gms.) of a good fish liver or dried yeast preparation. The vitamin requirements of growing children are relatively much more than those of adults and it is unwise to reduce the dose for age. In cases with physical signs of definite deficiency disease, this dosage can be doubled and repeated two or three times a day, until improvement sets in.

A standard fish liver oil should contain at least 600 International Units of vitamin A and 85 I.U. of vitamin D per gram. However Indian fishes, in comparison with European fishes, yield liver oils relatively very much richer in vitamin A than in vitamin D. Unless specially fortified with vitamin D, many preparations would be relatively poor in this vitamin. As however rickets (see Chapter 3) is uncommon in most parts of India, oil containing lower amounts of vitamin D can safely be used, *except* for a population amongst whom rickets is prevalent. A good dried yeast should contain at least vitamin B₁ 2.5 mgms., riboflavin 5.0 mgms. and nicotinic acid 15 mgms. per 100 gms. respectively. Yeast extracts such as marmite, vegemite and yex can also be used in suitable equivalent quantities.

Fish liver oils and dried yeasts should be readily available in sufficient quantities from Indian sources. At the moment their commercial production is not fully developed. Practitioners should remember that both products lend themselves readily to unscrupulous adulteration: it is recommended that only those made by firms with established reputations and facilities for check-

ing the vitamin content of their products be used. Two satisfactory preparations are "Shark Liver Oil" manufactured by the Government Oil Factory, Calicut and "Food Yeast" made by Messrs. Parry & Co., Ltd., Madras. Prices are not yet sufficiently low, but the cost should work out at a fraction of an anna per dose.

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CHAPTER 3

SPECIFIC DEFICIENCY DISEASES

BERIBERI

Definition. Beriberi is a disease characterised by multiple neuritis and myocardial failure with oedema. It is caused by a deficiency of vitamin B₁ in the diet. In India it is common only in the north eastern coastal districts of the Madras Presidency, where it is associated with the consumption of diets, in which "raw" highly milled rice is the principal source of calories.

Etiology. The essential role of vitamin B₁ deficiency in the causation of beriberi has become generally realised. There can now be no reason to doubt that a deficiency of this vitamin is an underlying essential factor in all cases of human beriberi. However, the disease in man is seldom so clear cut as the disease, which can be produced in experimental animals on a B₁ deficient diet, and it is probable that in individual cases other deficiencies, especially of vitamin A, and infections modify the clinical picture. It is now well established in experimental animals that vitamin B₁ plays an essential role as a co-enzyme in the intermediary metabolism of carbohydrates. In the absence of the vitamin B₁ in the diet a failure occurs in the normal mechanism for the utilisation of glucose by the tissues. The tissues are no longer able to metabolise pyruvic acid ($\text{CH}_3 \text{ CO COOH}$) which is a stage in the normal breakdown of carbohydrate. In consequence this acid accumulates in the tissues, notably in the brain and blood. The ability of the tissues of vitamin B₁ deficient animals to metabolise pyruvic acid can be restored by the addition of the pure vitamin in vitro. A similar metabolic effect occurs in acute human beriberi and pyruvic acid has been demonstrated in abnormal amounts in the blood of such patients. Both in the vitamin B₁ deficient animal and the acute case of human beriberi, there is a rapid clinical improvement and a corresponding disappearance of pyruvic acid from the blood following treatment with the vitamin. There can be little doubt that the same breakdown occurs in the fundamental processes in the enzyme chemistry of both and this demonstrates the primary importance of vitamin B₁ deficiency in the causation of human beriberi.

Diets deficient in vitamin B₁ are almost invariably deficient in other essential dietary factors and it is probable that some of the features of the human disease are referable to these deficiencies. This is a matter for further clinical research.

It is well-known that an attack of beriberi is frequently ushered in by some unusual physical disturbance, such as the performance of exceptionally arduous

labour, a severe febrile illness, pregnancy or an attack of diarrhoea. Whilst the latter probably acts by preventing the absorption of vitamin from the intestine, the other three factors increase metabolism and thereby increase the demands of the tissues for the vitamin. In this way they may precipitate an attack of beriberi.

Vitamin B₁ Requirements. The work of Williams and Spies (1938) has thrown considerable light on the problem of vitamin B₁ requirements. These workers studied the composition of a series of human diets reported to have produced beriberi or protected against the disease, and showed that the likelihood of any given diet leading to beriberi is influenced not only by its vitamin B₁ content but also by its caloric yield. This is in conformity with the hypothesis that vitamin B₁ is concerned in carbohydrate metabolism; the more abundantly carbohydrate—the chief source of calories in all diets based on cereals—is consumed, the greater the requirements of vitamin B₁. Williams and Spies calculated the vitamin B₁ and calorific value of a hundred diets, using reliable figures for the vitamin B₁ content of individual foods. They showed that the ratio vitamin B₁ content/calorific value can be used to predict the beriberi-producing effect of any particular diet. Thus, in 61 out of 66 diets associated with beriberi the above ratio was less than 0.250. Thirty one out of 34 diets not associated with beriberi gave values over 0.250. Out of the 100 diets studied only 8 gave discordant values and 4 of these were very close to the dividing line.

A number of workers have demonstrated that fat has “a vitamin B₁-sparing” action. Vitamin B₁ is not concerned in the metabolism of fat and hence the replacement of carbohydrate by fat in a diet reduces vitamin B₁ requirement. Williams and Spies consider that in America the relatively high fat content of the diet is an important factor in preventing the occurrence of beriberi in certain sections of the population, which consume large quantities of highly milled cereals. Accordingly, they suggest that the vitamin B₁/non-fat-calorie ratio is a more accurate measure of the adequacy of the vitamin B₁ content of a diet. In India the intake of fat by poor rice-eaters is usually very small, being often less than 15 gms. and rarely more than 30 gms. In considering vitamin B₁ requirements in India and the tropics generally, the fat content of the diet can probably be neglected without introducing any great error. The simpler vitamin/calorie ratio is sufficiently accurate and can be rapidly calculated.

An individual consuming a diet with an adverse vitamin/calorie ratio is *in danger* of beriberi; it does not necessarily follow that he will contract beriberi. Experience shows that the onset of beriberi in those consuming a vitamin B₁ deficient diet is usually brought about by some additional metabolic strain which increases the demand for vitamin B₁. Severe physical exertion, a feverish illness, particularly if accompanied by diarrhoea, and lactation are all likely to precipitate the disease.

For a diet with the calorific value of 2600, the amount of vitamin B₁ required to give a vitamin/calorie ratio of 0.25 is about 620 micrograms.* This may be taken as the minimum amount needed to prevent the danger of beriberi. It allows little or no margin of safety. Diets based on home-pounded or parboiled rice usually supply from 700 micrograms to one milligram.

Requirements of vitamin B₁ are influenced by a variety of factors. The amounts needed to prevent beriberi in individuals living on ordinary diets cannot be taken as representing the "optimum." First, emergencies such as fever, spells of exceptionally hard manual work, pregnancy, lactation and (we may perhaps add) alcoholic bouts have to be provided for. Thus, severe beriberi is very uncommon amongst parboiled rice-eaters in India, but mild peripheral neuritis, presumably related to vitamin B₁ deficiency, is not uncommon in lactating women living on parboiled rice diets—*i.e.*, such diets are not sufficiently rich in vitamin B₁ to meet *all* contingencies. Secondly, an intake 2.3 times above the usual minimum necessary to prevent beriberi may be physiologically advantageous and lead to a higher level of general health and a lowered susceptibility to disease. All things considered, a daily intake between 1.5 and 2.0 milligram, probably represents a suitable standard.

The likelihood of any rice diet producing beriberi, and the margin of safety provided, can be roughly assessed by a glance at a schedule which gives daily intake in terms of actual foods. Such diets usually contain large quantities of rice and much smaller amounts of pulses, vegetables, etc. Vegetables, fruit, milk and meat (except pork and glandular organs) are not rich sources of vitamin B₁. (See Appendix 1). They contain as a rule just sufficient vitamin for the metabolism of the calories which they yield, and when rice provides over 70 per cent of total calories have a negligible influence on the vitamin/calorie ratio. The adequacy of poor rice diets of the usual kind in vitamin B₁ content depends essentially on the method of preparation of the rice and the amount of pulse consumed.

Before going on to discuss the epidemiology of beriberi, a digression must be made here to consider briefly the effect of the various methods of preparation of rice on its vitamin B₁ content. Whole rice, like all other cereal food grains, is a rich source of the vitamin, containing about 3.4 micrograms per gram. But the majority of this is contained in the outer layers of the pericarp and in the germ and is subject to loss in the process of milling. If the milling is carried out by pounding in the home the majority of the pericarp of the grain is retained and such rices usually contain 2.3 micrograms of vitamin B₁ per gram. However, if the milling is carried out in a modern machine mill and if the grain is highly polished, the majority of the pericarp is removed

*1,000 Micrograms	= 1 Milligram (mgm)
1,000 Milligrams	= 1 Gram (gm)

and such rices usually have a vitamin B₁ content of less than 1.0 microgram and, in some cases, as much as eighty per cent of the vitamin may be removed by the millers.

Another important process that affects the vitamin content of rice is parboiling. "Parboiling" means the steaming or boiling of unhusked rice after soaking, but before milling. This splits the woody husk and makes its subsequent removal easier. Parboiled rice after steaming is pounded or milled in the same manner as raw (unsteamed rice). It has been shown that in the process of steaming the vitamin is driven into the grain and subsequently is much less readily removed by milling. Thus even highly polished samples of parboiled rice usually contain at least 2.0 micrograms of vitamin per gram.

After milling rice has to be washed and cooked before it can be eaten. Vitamin B₁ is very soluble in water and losses here may be large. Losses are of course variable and the less water used in the household, the smaller they will be and the more vitamin will be retained for human consumption. These changes are shown in Table 6.

TABLE 6

The effect of milling, washing and cooking on the vitamin B₁ content (microgram/gram) of rice.

Rice.	Uncooked.	Washed and Cooked	Vitamin Calorie
Raw home-pounded ...	2.4	1.0	0.30
Raw milled ...	0.9	0.3	0.09
Parboiled home-Pounded ...	2.4	1.1	0.33
Parboiled milled ...	2.6	1.1	0.33

The calorific value of one gram of rice is 3.3. It will be seen that the vitamin/calorie ratio of raw home-pounded, parboiled home-pounded and parboiled milled rice is about 0.30, a value just on the right side of the dividing line of 0.25. A diet consisting mainly of these rices is normally protective against beriberi, though the margin of safety is small, and prolonged washing of the rice, or the subjection of the person consuming it to some excessive metabolic strain may involve danger of beriberi. Raw milled rice often contains less than 0.5 micrograms of vitamin B₁ after washing and cooking. This gives a very low ratio and there is a large deficit, which unless it can be made up by supplementary foods involves a grave danger of beriberi.

With these observations in mind we are now in a position to consider the figures available of the distribution of beriberi in India.

Epidemiology. Beriberi occurs throughout the rice-eating countries of the East. In India, however, the extent of the disease relative to the total number

of rice-eaters has always been slight in comparison with that observed in Malaya, Indo-China, Siam, Japan and the Philippines. Although rice is the staple cereal of over half the population of India and beriberi is known to be a disease associated with ill-balanced rice diets, in most parts of the country typical beriberi is unknown, or only rare cases or small outbreaks have been reported. As an endemic disease it is limited to an area in the North East coast of the Madras Presidency, the Northern Circars—including the Ganjam, Vizagapatam, Godavari, Kistna, Guntur and Nellore districts. In other parts of the country only sporadic outbreaks occur.

TABLE 7
Beriberi in the Madras Presidency.

Northern Circars (Beriberi area) Population 13.9 million		Remainder of Madras Presidency. (Population 32.9 million)		
Year.	Cases.	Deaths.	Cases.	Deaths.
1932	16,091	93	665	3
1933	20,513	55	822	2
1934	21,849	42	711	5
1935	34,639	11	382	8
1936	20,814	91	452	7
1937	27,488	65	748	5
1938	32,717	64	649	4

The map shows the position of the affected districts in the Madras Presidency. Here the disease has been endemic for many years. Malcolmson (1835) recorded its presence in the Northern Circars over a hundred years ago. The incidence of beriberi in the Madras Presidency during recent years is shown by Table 7, based on the annual reports of the Madras Public Health Department. It will be seen that although the population of the "beriberi areas" is only 30% of the total population of the Madras Presidency, 95% of the cases of beriberi occur in these areas. The figures represent cases applying for medical treatment in hospitals and dispensaries and these give only a rough indication of the incidence of the disease.

In most Eastern countries the theory that beriberi is a deficiency disease caused by the use of rice decorticated by machine milling has been accepted

without question for many years. But in India itself there have been numerous controversies about the etiology of the disease. It has been maintained that beriberi is due to an infective organism or a toxin present in rice stored under bad conditions. An infective element, superimposed upon diet deficiency, has been postulated by some workers. Much of the confusion has been due to the lack of a clear cut relation between the use of machine-milled rice and beriberi. In Madras some 70 per cent of the rice-eating population consumes machine-milled rice and the curiously limited incidence of beriberi cannot be explained by a special preference for milled rice in the endemic areas. Another source of difficulty has been the confusion between beriberi and epidemic dropsy. There is no relation between the latter disease (See Chapter 5) and the kind of rice consumed.

The whole problem has been clarified by the discovery that parboiled rice remains relatively rich in vitamin B₁ after milling, owing to the diffusion of the vitamin inwards into the grain. The essential difference between the food habits of the people in the beriberi area in the Madras Presidency, and those followed in other rice-eating areas of India, is that in the former area paddy is usually milled in the raw state, as in other Eastern countries. In a diet survey of 16 families carried out in the Godavari delta on the east coast of Madras the following average figures (ozs. per consumption unit daily) were obtained:

Raw rice	...	...	...	...	20.5
Parboiled rice	...	...	...	...	1.4
Pulses	...	...	...	...	0.8
Leafy vegetables	...	...	...	...	0.4
Non-leafy vegetables	...	...	...	...	2.9
Fruits	...	...	...	...	0.2
Oils and fats	...	...	...	...	1.2
Fish, meat and eggs	...	...	...	...	0.7
Sugar	...	...	...	...	0.1
Buttermilk and curds	...	...	...	...	5.0

("Buttermilk" was usually well-diluted, and the total consumption of milk in terms of the whole product was small.)

This is the ordinary poor rice-eater's diet, based on *raw* rice as the staple. In the majority of families at the time of the survey the rice consumed was machine-milled. No survey of rice-eating rural groups outside the beriberi area has given similar results—in all cases parboiled rice was the chief ingredient in the diet. In Assam, Bihar, Orissa, the United Provinces, most parts of Bengal and the remainder of the Madras Presidency, parboiled rice is preferred by the majority of the population; this may be home-pounded, but much of it, particularly in Madras, is machine-milled. The superiority of parboiled rice in vitamin B₁ content appears to explain the epidemiology of beriberi in India.

In the Northern Circars, beriberi is essentially a disease of those, who consume machine-milled rice but it does occur occasionally when hand-pounded rice is used. Beriberi was recognised as a disease before the mechanical rice mill was invented (Malcolmson, 1835). The explanation is that it is possible, but not the usual practice, to remove the germ and pericarp almost as completely by home-pounding as by the machine mill. There is the further possibility that excessive washing may, on occasion, reduce the vitamin B₁ content of hand-pounded rice to a dangerous level. Unquestionably, however, the disease has been, and still is, predominantly associated with the consumption of raw machine-milled rice.

Although beriberi is usually associated with a rice diet, this is not always so. It may occur when any highly milled cereal forms an excessive proportion of the diet. Examples are available of the occurrence of beriberi in groups consuming highly milled wheat. The relation between the state of milling of cereals and beriberi is illustrated by the outbreak during the siege of the British and Indian forces in Kut-el-Amara by the Turks in 1916. The Indian troops all ate whole wheat chapatties and did not develop beriberi. The British troops consumed white bread made from refined wheat flour, and beriberi broke out amongst them. Although beriberi is at present unknown amongst the wheat-eating population of North India, a very real danger exists. There are already signs of an increasing popularity of white bread amongst all races of Indians. This was especially noticeable amongst the widely travelled sepoys. The widespread substitution of the indigenous chapatti made of wholemeal flour by bread made of refined highly milled flour is a possibility that might occur, especially in the big industrial cities. Such a change over would result in a serious deterioration of health in the workers and might lead to the occurrence of cases of beriberi in large numbers in districts hitherto immune.

Clinical Features. The incubation period is very variable and naturally depends on the degree of vitamin deficiency in the diet. The body can maintain no large store of the vitamin and symptoms attributable to deficiency may occur within less than two months on an unsatisfactory diet.

The onset is usually gradual although on occasions severe paralysis or cardiac failure may set in suddenly. There is a great variation in the severity of the symptoms from case to case. In large outbreaks, some patients are completely crippled, others may present evidence of advanced cardiac failure and deaths may occur, yet the majority of patients are not completely incapacitated. Common complaints amongst those, who still carry on at their duties, are a loss of appetite, a lack of energy and general lassitude, palpitation, breathlessness on exertion, tingling and numbness in the feet, tenderness of the calf muscles, a slight loss of power and a general unsteadiness of the gait. None of these symptoms are specific evidence of the presence of beriberi:

indeed, although these are all undoubtedly manifestations of vitamin B₁ deficiency, they are also early features of a great variety of both organic and functional diseases. Only after a complete physical examination of the patient and a study of the history, especially an account of the recent food consumption, can an accurate assessment be made. The dietary history is most important. Unless the patient states that for the previous two months a highly milled cereal (*e.g.*, white bread or highly polished raw rice) has been the main article of food, it is best to look elsewhere for a cause of the symptoms and to hold out little hope of improvement following vitamin therapy. In the presence of a poor dietary history or, if the patient belongs to a group amongst whom beriberi already exists, then these symptoms should be taken as danger signals and adequate dietary treatment instituted.

For convenience of description beriberi is usually divided in three types, dry, wet and infantile. There are however, no fundamental pathological distinctions between the types and a considerable overlap may occur.

Dry Beriberi. This type presents the features of a multiple neuritis. The lower limbs are always affected more than the upper and the cranial nerves are seldom involved. The characteristic complaints are burning and tingling in the lower limbs, aching and cramps in the muscles and a loss of power in the legs. Walking may become impossible without the aid of sticks and in very severe cases the patient is bed-ridden. On examination the motor loss is always more than the sensory loss. Areas of anæsthesia in which all senses are affected are common. The muscles of the lower limbs can be seen to be flabby and wasted. Diminution or absence of the ankle jerks is an early sign. In severe cases the knee jerk is also lost. There is an unsteadiness in the gait and Rhombergism may be present due to impairment of postural sensation. Foot drop and wrist drop may develop and lead to deformities, unless corrected. The cerebrospinal fluid presents no changes. A mild tachycardia is usually present.

Beriberi has no characteristic features that enable it to be distinguished from other types of multiple neuritis such as those due to arsenic, diphtheria, chronic alcoholism or diabetes. The past history of the patient and his dietary and other habits usually make the diagnosis easy. The involvement of the fauces and muscles of accommodation in the palsies following faucial diphtheria enable the distinction between diphtheritic neuritis and beriberi to be made in most cases without difficulty. However, jungle sores on the legs may become chronically infected with *C. diphtheriæ* and give rise to a neuritis clinically indistinguishable from beriberi.

Wet Beriberi. This type presents the features of right-sided cardiac failure with œdema. In mild cases, the œdema is confined to the feet and legs, but in severe cases it spreads upwards and involves the trunk, arms, neck and face. Serous effusions in the peritoneal, pleural and pericardial cavities occur.

The earliest cardiac manifestations are palpitation and tachycardia. Systolic murmurs and reduplication of the second sound are not uncommon. Dilatation of the heart especially to the right and venous engorgement with congestion and pulsation of the veins in the neck occurs. Changes in the electrocardiogram, such as an inversion of the T wave, are common. Œdema of the lungs and engorgement of the liver may follow. Acute cardiac failure is a constant danger. It may develop suddenly and terminate fatally within forty-eight hours.

The urine presents no abnormalities. There is no fever and no specific blood changes.

The diagnosis of wet beriberi is usually easy. It rests on a suitable dietary history and the exclusion of urinary changes and valvular abnormalities or other evidence of structural cardiac damage. Confirmation can usually be obtained by the prompt response to vitamin B₁ therapy.

Infantile Beriberi. This condition is not uncommon in the beriberi areas in India. It occurs in breast-fed infants usually between the third and sixth month of life. The clinical features differ somewhat from adult beriberi. That the condition is essentially a vitamin B₁ deficiency has been shown by the satisfactory results of vitamin therapy and the close association with signs of beriberi in the mother. The disease exists in an acute and chronic form.

In the acute form heart failure may set in abruptly: the infant may suddenly become cyanosed with dyspnoea and tachycardia and die in a few hours. The disease may show itself in an apparently healthy infant and death may occur without medical or other aid.

In Singapore one mother made the descriptive and dramatic statement that "her baby turned blue, sighed and died." In less acute cases screaming fits, colicky pains and vomiting may precede the evidence of acute failure. One very characteristic sign, usually encountered in only severe cases, is partial or complete aphonia. The infant's cry may be thin and almost inaudible and in some cases it may go through the motions of crying without any sound proceeding from the mouth.

In the chronic form a general failure of nutrition occurs with vomiting and diarrhoea. Œdema and paresis have been reported, but are much less common than in the adult form and are usually absent.

The diagnosis of infantile beriberi may require considerable clinical acumen. It is a possibility that must be frequently borne in mind. A dietary history and examination of the mother for early signs of the disease may provide the clue to the true nature of the baby's illness.

Morbid Anatomy. In fatal cases of wet beriberi, at post-mortem, evidence of right sided heart failure is usually present. There is marked œdema of the lungs and distension of the great veins. Serous effusions are usually present. Dry beriberi is not directly responsible for death and in fatal cases intercurrent infections, especially tuberculosis, are usually found. Changes in the myocardium and in the peripheral nerves have been described. These are by no means constant in beriberi and death may occur with no changes in the minute structure, which can be detected by modern histological methods. It is clear that in beriberi, as in other vitamin deficiencies, changes in function take place before any visible changes in structure.

The peripheral nerves may show Wallerian degeneration of the axis cylinders: there may be axonal degeneration of the neuron. The sheath of Schwann sometimes shows a nuclear multiplication and invasion by leucocytes. The nerves in the leg are most affected, but the vagus and phrenic nerves may also be involved. In long standing cases degenerations affecting the posterior columns, posterior spinal ganglia and anterior horn cells of the spinal cord have been found.

The heart may be increased in weight and the right ventricle enlarged. Microscopic examination of the muscle shows fragmentation and degenerative changes.

Treatment. Although vitamin B₁ deficiency is the primary etiological factor in beriberi, pure preparations of the vitamin have not proved as generally reliable in treatment, as might have been hoped.

In acute cardiac beriberi with œdema dramatic improvement follows intravenous injection of the vitamin. A normal dose is 10 mgm. which may be repeated two or three times at four hourly intervals. In moribund patients up to 50 mgm. may be given. Symptomatic improvement should soon be evident and within a few hours respiratory distress reduced. Within from 24 to 48 hours the breathing should be easier, the cyanosis gone, the action of the heart steadier and slower and diuresis sets in. In fact so certain is the remedy that, if within this period, no obvious improvement has occurred, doubt must exist, as to the correctness of the diagnosis.

Similar spectacular results follow the treatment of the acute forms of infantile beriberi. A dose up to 5 mgm. may be given by injection subcutaneously and repeated at four hourly intervals.

No such prompt result follows vitamin B₁ therapy for the relief of the manifestations of peripheral neuritis of dry beriberi. In this type of the disease progress is always slow and must be measured in weeks and months, rather than hours and days. The writer is not convinced that parenteral vitamin therapy accelerates recovery in anyway. The reason for this is probably that these cases

are usually of long standing and structural nerve degenerations have occurred, which cannot be remedied in a short time. These degenerations may be associated with other vitamin deficiencies notably vitamin A.

In all cases of beriberi the general principles of treatment laid down in the previous chapter should as far as possible be carried out. It may be again repeated that a good mixed diet is of more value than pure vitamins, and that its value will be greatly vitiated, unless intercurrent infections are simultaneously eradicated.

For minor cases attending as out-patients a good yeast preparation is, at the same time, the cheapest and most valuable medicine. An amount equivalent to one ounce of fresh yeast should be given daily. It is seldom practical to wean the breast-fed infant. Treatment must be given *via* the mother.

Severe cases of peripheral neuritis may require suitable splinting and a long period of re-education and rehabilitation in a physiotherapeutic department. It is important to keep such patients about and out of bed.

The prophylaxis against beriberi is in theory essentially easy. If we could prevent the over-milling of rice and wheat, we could eliminate the disease. In India and the Far East, if parboiling of rice became a universal custom, beriberi would cease to be an important public health problem, although it might still be occasionally seen as a curiosity. However social custom continues to demand fine white rice and bread and for technical reasons millers still prefer to supply this demand, and so beriberi continues to be a serious cause of ill-health in many parts of the East. The problem has psychological, sociological and economic aspects, and there are legal and trade view points to be considered. It is a subject for study falling within the scope of social medicine, the latest addition to the medical faculty.

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PELLAGRA

Definition. A deficiency disease, primarily due to the absence from the diet of the B₂ group of vitamins, especially nicotinic acid. It is characterised by changes in the alimentary tract, the skin and the nervous system. It is

common amongst maize-eaters, in whom large epidemics arise. Occasionally small outbreaks occur amongst groups subsisting on rice, wheat and other cereals.

Clinical Features. An account of these must be given before discussing the vexed problem of etiology and the distribution of the disease in India. The old mnemonic, "Dermatitis, Diarrhœa and Dementia", provides a useful summary of the main features of the disease and the systems mainly affected.

Skin Lesions. The first change is a simple erythema, closely resembling sunburn. The lesion is symmetrical and is distributed over the exposed surfaces of the body. The back of the hands, the forearms, the front of the legs, the back of the neck, the front of the chest, the cheeks and nose are usually involved. But the distribution is not constant and may depend on the clothing habits of the subject. The condition is aggravated by sunlight and so in northern climates frequently breaks out in the early spring and subsides in the winter, only to recur again with each succeeding spring.

As the disease progresses, the erythema fades and gives place to extensive areas of exfoliative dermatitis. Large areas of skin become thickened, dry, hard and inelastic. Desquamation occurs readily and pigmentation is marked. The affected areas are usually sharply demarcated from normal skin. Healing tends to occur in the centre of a patch following desquamation. Pink areas of new skin may be surrounded by densely pigmented hypertrophic edges. The pink new skin slowly becomes pigmented, but remains thin and atrophic, unless a relapse occurs later.

Alimentary Canal. The glossitis and diarrhœa, which are features of pellagra, appear identical with the conditions described in the previous chapter, as forming part of the syndrome of mixed deficiencies. There are no signs or symptoms of disease of the alimentary tract peculiar to pellagra alone.

Nervous System. Mental symptoms are common: in mild cases, a general apathy and dullness are present. In more severe types a manic-depressive psychosis, sometimes associated with hallucinations is found. In long standing cases, this may develop into a complete dementia from which there is no recovery. A low grade peripheral neuritis, which is probably in reality a mild beriberi, is a frequent accompaniment.

Pellagra is a chronic disease that tends to recur year after year. It is not usually associated with a high mortality. Death when it occurs is generally due to progressive weakness and emaciation secondary to the diarrhœa or to intercurrent infection.

There are no definite specific pathological changes in the organs.

Treatment. The acute manifestations of the disease are in most cases promptly relieved by nicotinic acid. Nicotinic acid may be given either by mouth or subcutaneously. A maximal dose is 100 mgm., which can be repeated four hourly. If nicotinic acid is not available, yeast in amounts up to one ounce daily may be given. Improvement will usually be obvious within 48 hours, though complete recovery may take many weeks. The general dietary treatment necessary for all deficiency diseases is essential during convalescence. Special care must be taken to eradicate any intestinal worms that may be present.

Pellagra never occurs, if an adequate supply of protective foods, such as meat, vegetables and milk are available. Amongst maize-eaters, it can only be prevented by an all round improvement in the diet.

Etiology and Epidemiology. There is no question that pellagra is a deficiency disease and that it is associated with a lack of vitamins of the B₃ group in the diet. The disease only arises amongst persons living on diets containing inadequate amounts of nicotinic acid and the majority of the symptoms can be relieved by nicotinic acid. Nevertheless the disease is more than a simple nicotinic acid deficiency. The close association with maize consumption cannot be accounted for on these lines. For although poor maize diets contain insufficient quantities of nicotinic acid, they are no more lacking in this substance than poor rice diets. Yet true pellagra is uncommon amongst the rice eating population of India.

Many of the features of malnutrition seen in India are common to pellagra. In particular the glossitis and diarrhoea appear indistinguishable. In addition the skin changes, so common in deficiency states in India, bear some resemblance, especially as the hyperpigmentation and hyperkeratosis in these conditions are usually more severe on the exposed surfaces of the body. But the extensive exfoliative dermatitis and the extreme photo-sensitiveness are rarely seen. It has been thought that the mouth lesions and diarrhoea, so common in India were pre-pellagrous in origin. However, although glossitis and nutritional diarrhoea are so common and often so severe in rice-eaters as to be responsible for many deaths, yet true pellagra is rare. That it can occur must be accepted. Examination of photos of cases observed by Raman (1940) amongst rice-eaters in Vizagapatam leaves no room for doubt that they are pellagra. But numerous cases, which have been shown to the writer in various parts of India diagnosed as pellagra, have borne little resemblance to the florid cases, which are seen in maize-eating countries like Egypt. The extreme and characteristic photosensitiveness seems to appear only in maize-eaters. Why the poor rice-eater, who suffers from nicotinic acid and other B₃ deficiencies, so rarely gets true pellagra is unknown. It cannot be explained by differences in the content of known food factors in the two cereals. Pellagra is more than a simple nicotinic acid deficiency. Maize must differ from rice, either in the lack

of a hitherto unrecognised dietary factor or in the possession of a toxic component with the property of photosensitising the skin. True pellagra occurs amongst maize-eaters in India and Bajaj (1939) has given an account of the disease in the Kangra Valley.

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SCURVY

Definition. Scurvy is a deficiency disease due to the lack of vitamin C in the diet and characterised by hæmorrhages into the skin, subcutaneous tissues and mucous membranes.

Etiology and Epidemiology. Scurvy occurs whenever an insufficiency of vitamin C arises in the diet. Vitamin C is widely distributed in all fruits and vegetables and only very small amounts are required to prevent scurvy. Thus population groups have been observed receiving daily less than 5 mgm. of vitamin per head, in whom scurvy has not arisen. Although small amounts of vitamin C are capable of preventing overt signs of deficiency disease, there is considerable evidence that much larger quantities are necessary to maintain optimum health. It will be seen that the daily requirement given in Appendix 2 is 50 mgm. This is more than ten times the minimum required to prevent scurvy.

Scurvy is not a common disease in India. It is rarely seen, except in times of famine and even then only occasional cases occur. The writer has only seen a few cases in Hissar District of the Punjab during a famine in 1939. In numerous visits to famine camps both in Bengal and Madras, no single case was seen.

In the official reports on the Madras Famines at the end of the nineteenth century, there are numerous reports and discussions about the incidence of scurvy. In every instance the diagnosis appears to have been made on the presence of hæmorrhages from the gums. In view of the widespread incidence of severe pyorrhœa amongst Indians and the associated ease with which the gums bleed, it is doubtful whether a diagnosis of scurvy on this feature alone, in the absence of evidence of hæmorrhages elsewhere in the body, is justified.

Clinical Features. Scurvy is usually a disease of insidious onset and from four to eight months on a scorbutic diet may elapse before it sets in. Prodromal symptoms are apathy and general lassitude, with which pallor and a low grade anæmia are generally associated. These symptoms may precede by a long time the characteristic signs. Of these, the most striking though not always present,

are the changes in the mouth. The gums begin to swell, especially round stumps or carious teeth, and great hypertrophy may develop, so that the teeth become surrounded by a soft mass of tissue of bluish or purple tint. Bleeding is common and ulceration readily occurs associated with much foulness of breath. Equally characteristic are hæmorrhages into the skin and subcutaneous tissues—petechiæ and ecchymoses. The petechiæ are small, red or purple spots, which appear within the skin and remain usually about a week. They may coalesce into large areas. In severe cases they break down and form foul smelling discharging ulcers. Ecchymoses are produced by hæmorrhage into subcutaneous or intermuscular tissue. They may occur spontaneously or as result of minor injuries. They are commonest on the legs and may form quite large swellings, which are tender, brawny and pit on pressure. The skin over them is red, shiny and hot. Subperiosteal hæmorrhages, especially over the front of the tibia, are common. Hæmorrhages from the mucous membranes occur and bleeding may arise from the nose, mouth and rectum. Internal hæmorrhages are uncommon. The disease is usually associated with other signs of dietary deficiency, especially night-blindness. Anæmia is a constant accompaniment.

Scurvy may develop at all ages. At one time infantile scurvy, which usually develops between the ages of 8—10 months was frequently seen in Europe. The lack of vitamin is attributable to the destruction of vitamin C by heating milk and milk preparations given to bottle-fed infants. It is very rare in breast-fed children and for this reason is uncommon in India. The children affected are usually well-covered with flesh. After a few days of fretfulness, the prominent features appear. The most striking of these is tenderness of the legs, which causes the child to scream when touched or even at the approach of the doctor. The child lies very still, usually on his back with one or both legs everted and motionless. Examination usually reveals some swelling of the bones, most commonly in the lower end of the femur or upper end of the tibia. At the site of the swellings tenderness is acute and the skin over them is often tense and glossy and may be slightly œdematous. Hæmorrhages into the skin and subcutaneous tissues are much less common than in adult cases. Swelling of the gums round recently erupted teeth can usually be seen.

Diagnosis. This is easy in a well-marked case, provided the leading features of the disease are known. Adult scurvy may be mistaken for idiopathic purpura or acute lymphatic leukæmia. The infantile form may simulate acute osteomyelitis, syphilitic epiphysitis, rheumatic fever or infantile paralysis. However, the age incidence of these two infections is later than that usually found in infantile scurvy. An adequate dietary history will usually clear up any cases of doubt. Although the physician in India is unlikely to see any large outbreaks of scurvy, he may see isolated cases, which arise not uncommonly amongst

persons living and feeding alone and amongst food fadists. The only case seen by the writer, when a medical student in London, was in a man, living alone, who had been advised by his doctor to go on a milk diet. A rigid and exclusive adherence to the medical advice had been followed for nine months, and well developed scurvy had been the result. In these types of cases, which have been called "bachelor" scurvy, the history always gives the clue to the real cause of the condition.

Treatment. The acute manifestations of scurvy respond promptly to treatment with vitamin C. Pure ascorbic acid tablets should be given in a total daily dosage of 1000 mgm. for the first three or four days. Thereafter the dose may be cut down to 100 mgm. a day until convalescence is complete. For infants a dose of 100 mgm. a day is usually adequate.

Prophylaxis against scurvy is important. The bottle-fed infant should always be given some form of vitamin C daily. Orange juice is the most suitable, but tomato or any other fruit juice will suffice. The juice must be strained through fine muslin before it is given. The usual dose is one to three teaspoonfuls daily.

In time of famine or whenever there is a grave shortage of fresh vegetables or fruit, the germination of pulses provides a valuable additional source of vitamin C. The method which has proved satisfactory in the army is as follows:

"A sufficient quantity of whole (unsplit) dal or gram (say $1\frac{1}{2}$ to 2 ozs. per man) is soaked in water for 12 to 24 hours. A container big enough and holding sufficient water to allow for expansion should be used. Then pour off the water, remove the grains and spread on a damp blanket in a layer thin enough to allow access of air, and cover with another damp blanket. Keep the blankets damp by sprinkling with water. In a few hours small shoots will appear, and when these are $\frac{1}{2}$ to 1 inch long the process is complete. Vitamin C content is maximal after about 30 hours of germination." (*Field Service Hygiene Notes, India, 1945*). Pulses normally contain little or no vitamin C. One ounce of dried pulse may on germination yield 9-15 mgm. of the vitamin, an amount sufficient to prevent the appearance of scurvy. Distribution of germinated pulses have been widely practised in famine camps in India and there is reason to believe that it is a valuable measure for maintaining the health of the people in times of stress.

RICKETS AND OSTEOMALACIA

Definition. Rickets is a deficiency disease, due to absence of vitamin D from the diet, occurring in late infancy and early childhood, characterised mainly by changes in the bones leading to deformities. Osteomalacia is a deficiency due to lack of vitamin D and also calcium, occurring in later child-

hood and adult life and causing softening of the bones, leading to bending and deformities. Essentially rickets and osteomalacia are due to faulty diet, bad housing and social conditions, which limit access to sunlight.

Etiology. Both clinical experience and experiments on laboratory animals have shown beyond any doubt that rickets is attributable to a lack of vitamin D. This vitamin must be provided in the diet or, alternatively, it can be synthesised in the body by the action of ultraviolet light on precursors, which are present in the skin. A deficiency of vitamin D may be aggravated by a deficiency of calcium and phosphorus in the diet or by the presence in the diet of substances preventing the absorption of these elements from the intestines. A high carbohydrate diet has long been known to aggravate rickets. This has now been shown to be due to the presence in cereals (especially oatmeal and whole wheat and to a less extent in white flour and rice) of substances, which keep the calcium and phosphorus out of solution in the small intestine and so prevent their absorption into the blood. Phytic acid is the substance mainly responsible for this action.

Vegetable foods are, as a rule, very poor sources of vitamin D. Vegetable oils, green vegetables, fruits and cereals contain insignificant amounts. Animal sources are egg yolk, milk and milk fats, such as butter, cream and ghee. All animals store the vitamin in the liver and liver oils are a rich, but variable source of the vitamin. Fish liver oils, such as cod and halibut, have for long been known as valuable anti-rachitic agents and rich in vitamin D. Recently it has been shown that numerous Indian fish liver oils, notably the shark are equally effective and in some cases richer sources of the vitamin.

Epidemiology. (a) *Geographical Distribution in India:* Rickets and osteomalacia are widespread in Northern India. These diseases are particularly common in the larger cities such as Delhi, Amritsar and Lahore. In Calcutta and Bombay they are less prevalent. Outside the cities, there are two areas in North India, in which rickets and osteomalacia are known to be very serious public health problems—the Kangra valley in the Punjab and Kashmir. The Punjab Health Department (1939) has recorded that of 110 women examined in 4 villages in the Kangra valley, 46 showed signs of osteomalacia. Wilson found that 28 out of 38 families investigated in the same area contained cases of osteomalacia.

Vaughan (1926) has reported that “almost all Kashmir women who have borne children are affected in some degree, with the exception of the boat-man class.” The women of this class live in the open air in the large boats on the Jhelum river. The worst cases are seen in the houses of the wealthy, who keep their women in seclusion and among those of the poorer classes following the same practice. In Kashmir osteomalacia in its acute stages is much more

common in winter and spring, during and after confinement to the house because of cold weather, than at other seasons.

Wilson examined 1482 school girls in Lahore for rickets. She found signs in 43 per cent. In one Mohammedan school 33 children in a class of 55, with ages ranging from 5 to 10 years showed evidence of the disease. Its incidence among infants in Northern India requires further investigation.

By contrast South and Central India are relatively free from rachitic diseases. Many thousands of children in South may be examined, before encountering a case of outspoken rickets. Osteomalacia is rare among Madrassi women. Some indication of the geographical incidence of osteomalacia is provided by the number of cases of contracted pelvis encountered in hospitals in various areas, osteomalacia being the commonest cause of obstructed labour. The following figures are given in the Report of the Central Advisory Board of Health (1938) on maternity and child welfare work in India.

	<i>Contracted pelvis.</i>
Lady Hardinge Hospital, New Delhi	... 1 in 11 deliveries.
Agra Maternity Hospital (United Provinces)	... 1 in 15 ..
Cama Hospital, Bombay	... 1 in 35 ..
Caste and Gosha Hospital, Madras ...	... 1 in 90 ..

In general the incidence of contracted pelvis decreases, *pari passu* with the incidence of osteomalacia, from North to South.

(b) *Sex*: The incidence of juvenile rickets is approximately the same in both sexes. Osteomalacia, however, is essentially a disease of women. Tetany and some of the earlier signs such as pain in the bones may occur in males, but actual bone deformities are not common.

(c) *Social and religious factors*: Osteomalacia is very closely associated with the purdah system. The disease in Northern cities is relatively more common in Mohammedan women, who keep strict purdah. Many victims of osteomalacia enter strict purdah at the age of ten and after this never leave their homes without being heavily clothed and completely veiled. Among the Hindus, the victims of osteomalacia often belong to the families of money-lenders, shop-keepers and small merchants. The women of these communities, though they do not keep strict purdah, leave their houses rarely. The younger female members of the family, who belong to the age groups which suffer most from osteomalacia go out of doors even less frequently than the older women. Vaughan (1926) shows how the frequency of osteomalacia in various districts and towns in North India depends on the seclusion or otherwise of the women.

These points are illustrated by figures given by Balfour (1927). The percentage incidence of osteomalacia in Hindu, Mohammedan and Indian Christian women in maternity hospitals in various parts of India was as follows:

TABLE 8

Osteomalacia in different communities.

1. Hospitals in various cities.

Community.	Total No. of labour cases.	No. of cases of osteomalacia	Ratio per 1000 labour cases
Hindu ...	5167	160	30.9
Mohammedan ...	1273	79	62.6
Others (mainly Indian Christian)	1152	6	5.2

2. A Bombay Hospital.

Hindu ...	2066	6	2.9
Mohammedan ...	842	32	38.0
Others (mainly Indian Christian)	801	Nil	Nil

The predonderance of Mohammedan women suffering from osteomalacia is striking. Hindu women in Bombay contract the disease more rarely than Hindu women in Northern India; the former lead a more active life and do not confine themselves to the house. Christian women, who have a higher standard of education, mix normally in the life of the community. "Literacy" Balfour says "is more advanced in the Christian than in the other communities. Moreover, it is a loose term. With Hindu and Mohammedan women it too often means only a year or two at a primary school. With Christian women it often means six or eight years at a mission school where the girls are trained in hygiene and physical exercises as well as in book-work. Such a girl in her after life will be healthier, wiser as regards food and more ready to seek advice in illness."

(d) *Housing*: Bad housing conditions are an important factor in the causation of rickets and osteomalacia. Rachitic diseases flourish in the ancient parts of Northern Indian cities with narrow winding streets, flanked by dark, damp, ill-ventilated and over-crowded houses. Houses are often built round a central courtyard into which the sun rarely penetrates. Such conditions, combined with the purdah system and the sedentary habits of the Hindu women, effectively prevent the provision of vitamin D through the action of sunlight on the skin.

In the Kangra Valley in the Himalayan foot hills, rickets and osteomalacia are prevalent in a rural population. Women who work in the fields are often

affected. Clothing is usually somewhat heavy and for climatic and geographical reasons there is less strong direct sunlight available than in Central and Southern India. The pains of osteomalacia often come on during the rainy season when the sky is over-clouded for several months.

(e) *Diet*: Descriptions of the kind of diet eaten by victims of osteomalacia have been given by various writers. In general the diet is largely composed of wheat or rice, or a mixture of both, with some pulses and a minimum of milk products, vegetables and meat. Essentially it is a diet deficient in vitamin D and calcium. Some of the wealthier patients may consume some milk fat (ghee), but probably not in large amounts. Usually vegetable oil is used in cooking. Mohammedan women of the more prosperous classes eat some meat. Hindu women of the same economic status are nearly all vegetarians.

More exact information is available about the diet of various villages in the Kangra Valley. The Punjab Public Health Department (1939) studied the diet of 15 families in the village of Launa, in which the incidence of osteomalacia is very high. The intake of various foods per consumption unit per day was as follows:

						Ozs. daily.
Rice	...	...	...	...	...	14.4
Wheat	...	...	...	...	...	7.4
Maize	...	...	...	...	...	1.1
Pulses	...	...	...	...	...	0.9
Leafy vegetables	...	...	...	...	...	0.4
Non-leafy vegetables	...	...	...	...	...	0.7
Fruit	...	...	...	...	...	none
Meat	...	...	...	...	...	0.1
Milk and butter-milk	...	...	...	...	...	0.8
Ghee	...	...	...	...	...	none
Vegetable oils	...	...	...	...	...	0.1
Sugar	...	...	...	...	...	0.1

The defects of this diet are obvious. It contains almost no vitamin D and provides only about 0.2 gramme of calcium daily. Wilson observed that within this particular village the incidence of osteomalacia varied with the nature of the diet. "Those living on cereals and practically without milk, eggs, greens or meat showed a heavy incidence; those better off and having a little milk and perhaps a slightly better diet, a milder incidence; those having milk, occasionally fresh greens and fruit and more cooked vegetables and meat, showed no signs of the disease."

Pathology. The exact mechanism of the action of vitamin D is not known. There is some evidence that it promotes absorption of calcium from the small

intestines, but more probably its main function is locally in the growing bone, where it is considered to be primarily concerned with the precipitation of calcium phosphates out of solution in the plasma into an insoluble form.

The principal morbid signs are a softening of the bones, a slowing of bony growth and an enlargement of the epiphyseal cartilages. As a result of the softening, the bones bend readily and the characteristic deformities of the disease arise. Histologically the appearance of the epiphyses has been described as an excessive preparation for ossification, but an inadequate performance. There is an excessive multiplication of cartilage cells in the epiphyses and a great increase in vascularity with a multiplication of capillary loops. Hence the epiphyses are actually larger than normal. But the cartilage cells are not arranged in regular columns as in normal bone formation; in consequence calcification is deficient and proceeds irregularly. The periosteal covering of the bones is more vascular than normal and the distribution of the vessels irregular.

Clinical Description. (a) *Rickets:* A typical case is recorded, as described by Dr. C. F. Still, a great pædiatrician of a past generation.

“An infant aged about eighteen months is brought for inability to walk. He has been suckled entirely for six or eight months, and then has had condensed milk and occasionally bread and potato. For some months past, he has sweated profusely about the head and neck whenever he falls asleep. Dentition did not begin until he was nearly a year old, and even now he has but three or four teeth. He has never walked, and as he sits on his mother's lap there is a marked kyphosis, especially in the upper lumbar region. He is pale and fretful; perhaps rather fat but flabby; the head is unduly large, with a tendency to spareness; the anterior fontanelle is much more widely open than it should be at his age. The chest is ill-shaped, the sternum a little prominent, and there is some transverse constriction below the nipples. A row of knob-like eminences marks the junction of the costal cartilages with the ribs on each side of the chest; the lower ribs are somewhat everted above the big abdomen, in which the liver can easily be felt and the spleen is also just palpable. The muscles feel flabby and soft. Just above the ankle and the wrist, the bones seem thickened. There is little or no abnormal curvatures of the bones. Such is a moderate degree of rickets.”

There is little to add to this precise description. It may be emphasised that rickets is a disorder of nutrition and as such it affects the whole body. The symptoms are such that it may be suspected (and prevented) before definite physical signs and bony changes appear. The disease first makes its appearance at the end of the first year of life. Then a flabby baby, unable to pull itself up, fretful and easily irritated, without teeth, liable to profuse sweats, should arouse suspicion. Such a child ought to have vitamin D therapy, *before* bony changes occur.

Severe cases are often associated with nervous symptoms. Fretfulness has already been noted. Convulsions may occur and are frequently associated with rickets in young children. The central cortex becomes in some way unstable and any trivial illness or physical upset may bring them on. True tetany may arise in rickets. A tonic spasm occurs, which affects chiefly the muscles of the forearm, hand, leg and foot. The hand is generally slightly bent at the wrist, the thumb is adducted and pointing to the interval between the ring and middle fingers and the fingers are semi-flexed at the metacarpo-phalangeal joints and extended at the phalangeal joints, often with some tendency to over extension at the latter. The toes may be crowded together in a similar position. The spasm in some cases persists for several hours: in others it is intermittent lasting only a few minutes. It is associated with facial irritability and gentle tapping over the superficial branches of the facial nerve may give rise to contractions of the muscles supplied. Tetany assumes a specially dangerous form when spasm of the larynx (*laryngismus stridulus*) occurs. The spasm may suddenly close the glottis so that the infant for a few seconds is unable to take in breath: the face becomes livid and the infant looks terrified and makes violent efforts to inspire. After a few seconds the spasm relaxes and a deep inspiration is taken with a loud cooing noise. These attacks may come on many times in the day and have on occasion caused death from asphyxia.

The nervous manifestations of rickets are due to an incidental fall in the serum calcium levels, which may be as low as 5 to 7 mgm. per 100 ml. of serum. The normal level is 10-12 mgm. This fall is not constant and in most cases serum calcium levels stay within the normal range. A more constant blood change is in the inorganic phosphorus content, which usually falls from the normal level of 5 mgm. per 100 ml. of plasma to 2 mgm. The enzyme phosphatase, which is responsible for the splitting of phosphorus compounds and the deposition of calcium phosphate in bone, is also present in increased amounts in the blood.

Actual deformities do not appear until the child has been moving about and the soft long bones have had to bear weight. The commonest deformity is a curving forward of the tibia with the concavity on the inner and posterior surface giving rise to characteristic bow legs. Curving of the femur may also take place and kyphosis of the spine. Anterior posterior flattening of the pelvis is an important deformity in females. Other less common deformities are coxa vara, knock knees, curvatures of the bones of the arms and permanent dwarfing.

Characteristic changes in the x-ray appearances at the ends of the long bones occur. In the normal growing bone the end of the diaphysis is smooth and sharp and there is a clear space between it and the epiphysis. In rickets the end of the diaphysis is irregular and uneven: the end of the growing bone appears broadened, hollowed and ragged. These changes are often most marked

at the wrist and may be useful both for diagnostic purposes and for following the course of healing. A generalised osteoporosis may also be present.

Rickets is associated with an increased susceptibility to infection, especially of the respiratory tract. Bronchitis and broncho-pneumonia are common accompaniments and frequently have a fatal result. In this manner rickets may be indirectly the cause of a large mortality, although the disease is seldom the direct cause of death.

(b) *Osteomalacia*. The manifestations of deficiencies of Vitamin D and calcium in the diet in adults are slightly different than in infants and children. In the adult softening of bone (osteomalacia) occurs giving rise to gross deformities. The pelvis, in particular, is affected and difficulties in labour result, which are responsible in part for the high maternal death rate in the big cities of North India. The anatomical nature of the deformities and their effect on the normal female passages are not within the present scope and obstetrics text-books should be consulted. But it is now realised that early signs and symptoms arise before marked skeletal changes have developed. It is at this stage that treatment is effective. Bones once twisted cannot be restored to their original shape. Dr. D. C. Wilson (1929) has made a careful study of these manifestations and gives prominence to the following points in the early stages of the disease:

- (1) An uneven gait on a slightly wide base. The patient shows a tendency to swing the leg outwards before putting the foot to the ground.
- (2) Tetany and hyperexcitability of the muscles with exaggerated knee jerks. Carpo-pedal spasm and facial twitching may occur.
- (3) The pelvis from behind may appear flattened, wider than normal and slightly tilted.
- (4) *Pain*. This ranges from a dull ache to pain of severe degree. Common sites are over the sacrum and lower lumbar vertebræ, or the bones of the pelvis and the long bones. Pain in the ribs may also be severe.
- (5) Tenderness on pressure is common at many bony prominences. It is especially marked over the subcutaneous surface of the tibiæ.
- (6) Spontaneous fractures may occur.

These signs and symptoms precede the deformity of the pelvis and may be mistaken for muscular rheumatism. Some degree of anæmia is almost invariably present and menstruation is usually scanty. Very often the onset coincides with

the first pregnancy, a remission occurring after delivery and a return of symptoms in a more severe degree in succeeding pregnancies.

The x-ray appearances show that apart from the deformities and fractures, a generalised osteoporosis takes place. The cortex of the bones becomes thinner as the disease advances, the process progressing from within outwards. In some cases it can ultimately be distinguished only as an attenuated covering to the bones.

As in rickets changes in the blood chemistry arise. If the normal limits for serum calcium and serum inorganic phosphorus are taken to be 9-11 and 2-5 mgm. per 100 ml. respectively, the values observed in many cases of osteomalacia fall below the normal. But in many mild cases the values are within the normal range and it is clear that very low values are usually found only in severe and advanced cases. In the early stages of the disease the withdrawal of calcium and phosphorus from the bones maintains the content of the blood at its normal level.

As in rickets there is an increased concentration of phosphatase in the blood and determination of serum phosphatase may be of considerable value in the early diagnosis of osteomalacia.

Treatment. The treatment of rickets consists in providing a suitable supply of vitamin D. The daily dose required for cure varies from 1,000 to 3,000 International Units. The British Pharmacopœia Standard for Cod liver Oil is 100 I. U. per gram. The daily dose of such an oil varies from one-third to one ounce. Indian Shark-liver and Saw-fish liver oils are usually a little richer (about 1.5 times) than cod liver oil in vitamin D. These oils are, however, 8 to 10 times richer in vitamin A than cod liver oil. Accordingly they are sometimes diluted with vegetable oils containing no vitamin, to reduce them to approximately the same standard potency of vitamin A, as standard cod-liver oil preparations. Such preparations, of course, will be very poor sources of vitamin D.

Various concentrated preparations of vitamin D are on the market. Halibut oil is much richer than cod-liver oil and may contain 3,000 I. U. per gram. There are various preparations containing pure vitamin D as calciferol. Burroughs and Wellcome manufacture "Calciferol", a preparation containing 10,000 I. U. per "tabloid": Glaxo has an "Ostelin" preparation with 5,000 I. U. per ml. and "adexolin" capsules containing 1,000 I. U. per capsule.

With these concentrated preparations daily dosage is not necessary and the vitamin need be given only once or twice a week provided the average daily dose is maintained.

Vitamin D in large doses is toxic: a real danger of poisoning exists, especially if the potent concentrates are used without care. Several fatal cases have been reported, especially in infants. The symptoms of human vitamin D intoxication do not as yet appear to be well-defined. Such non-specific features as loss of appetite and loss of weight, nausea, vomiting and diarrhoea are described and should be warning signals. In experimental animals, pathological calcification of the soft tissues, especially the kidneys and large arteries can be produced.

Great care should be taken in the choice of a vitamin D preparation. Only those made up by firms of high repute should be used. General agreement as to the optimum dosage has not yet been reached and the manufacturer's instructions as to dosage should be followed rigidly. The writer knows of no concentrate of Indian manufacture. Vitamin D preparations, like those of vitamin A, lend themselves readily to adulteration with inactive vegetable oils.

Sunlight, either natural or artificial, is a valuable additional form of treatment. Exposures should at first be short, say ten minutes twice a day, and gradually extended as tolerance is developed.

An all round improvement in the diet is necessary. An increase in eggs, milk and milk products, the principal foods containing the vitamin is particularly desirable. If possible the amount of cereal in the diet should be reduced, fats, meat or vegetables being substituted in suitable proportions.

Tetany is the direct result of calcium deficiency and can be cured by calcium therapy. Milk is the best and most readily absorbed source of calcium. The amount may be increased by the addition of calcium chloride 15 grains three times a day. This should not be continued for more than two days as a real danger of acidosis exists. In severe cases if convulsions are continuous, calcium can be given intravenously. Calcium gluconate (10 ml. of a 10 per cent solution) or other suitable preparation may be used. If convulsions are frequent a sedative is required. Of homely remedies a warm bath is the best and of drugs, chloral hydrate in a dosage of one grain for the one year old child is satisfactory.

To correct deformities the above treatment must be supplemented by rest which must in severe cases be in bed. When no exercise is being taken, massage is a valuable aid. In a growing child the prognosis of deformities is good and, provided suitable rest and dietary measures are provided, limbs will straighten in a remarkable way. In older children there is less chance for natural correction to take place and an orthopædic surgeon may have to be consulted.

Osteomalacia has the same fundamental pathology as rickets and the treatment is essentially the same—vitamin D and calcium. Cod-liver oil should be

given in quantities of one to two ounces a day or equivalent amounts of other vitamin D preparations. An all round dietary improvement with increased milk consumption is desirable, if practical. Extra calcium can be given as in rickets. Sunlight is also valuable. Experience in India suggests that the best results can only be obtained by a combination of all these methods.

Prevention. The public health problem presented by rachitic diseases in Northern India is a formidable one. There are two distinct factors in causation, faulty diet and insufficient sunlight and in any preventive campaign the attack must be aimed in two directions. "More sunlight" as far as most of Northern India is concerned, means first of all the breaking down of ancient social and religious prejudices. The purdah system originated in times of disorder, massacre and rape; in a modern peaceful community it is as much as anarchism as a medieval walled city. Every step towards the abolition of purdah is a step towards the abolition of osteomalacia. But, as has been pointed out, Hindu women who are not strictly in purdah may be confined to the house almost to the same degree as their Mohammedan sisters. Ignorance is one of the fundamental causes of osteomalacia. The raising of the standard of education and the community generally is essential if the disease is to be eliminated.

Among the wealthy, who can afford houses with interior gardens for their women folk, the effect of seclusion is likely to be relatively slight. Good housing conditions will do much to prevent osteomalacia. The actual housing conditions in Indian cities are intolerably bad; the foulness of the slums, to which too little publicity is given, is appalling. Rickets and osteomalacia flourish in small dark dirty rooms crowded with human beings. The eradication of slums must be regarded as a necessary part of the campaign against rachitic diseases.

The improvement of diet presents great difficulties. The education of the population, and in particular the education of women, about food and its relation to health, is of essential importance. But when poverty is extreme and food supply deficient, education can achieve little. The Punjab Health Department (1939) points out that fundamental measures are needed for raising dietary standards and the prevention of rachitic and other deficiency diseases in the Kangra Valley.

"The ultimate reason for the present deficiency of milk, vegetables and fruits is in all probability the demands which both animals and human beings make upon the available resources of the soil. Heavy rainfall yearly depletes the soil of valuable soluble nitrogenous and other matter required for the production of crops. The replenishment of the soil necessitates the maintenance, according to the local inhabitants, of more animals than are required for labour and other purposes in order that sufficient quantities of manure may be available.

It would seem that the first necessity is to devise means to arrest exhaustion of the soil and improve its productivity. This may prove to be in large measure a question of afforestation.

Much waste animal and vegetable matter in villages is either never made use of as manure, or if it be used, it is not used in the proper manner."

The distribution of substances and preparations rich in vitamin D is a preventive measure of value under certain circumstances. Its efficacy largely depends on the existence of organisations in contact with the people. Maternity and child welfare institutions can provide anti-rachitic agents to pregnant women and such women should be persuaded to seek ante-natal advice and treatment. In the same way rickets can be prevented in infants and young children who are under the supervision of welfare centres. Certain mission hospitals have carried out effective preventive work along these lines after the confidence of the people has been gained and the habit of applying readily for medical advice and treatment established. Women suffering from the pains of osteomalacia in the early stages of pregnancy recognise the cause of their troubles and seek treatment and ante-natal supervision reduces the dangers of labour.

The distribution of anti-rachitic agents on a wider scale should be possible. Fish liver oils are beginning to be prepared in India on an increasing scale. Education and efficient "salesmanship" are needed by Health Departments so that the value of these products can be got across.

The causes of rachitic diseases in India are now fully understood and their prevention is purely a public health and social problem.

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CHAPTER 4

ANÆMIA IN RELATION TO NUTRITION

Anæmia is not in itself a disease. It should be considered as a defect of the blood, which may arise from numerous disease processes. In India, it is extremely common and responsible for much ill-health and debility, especially in the child-bearing section of the community. Broadly speaking there are four main causes, which among them are responsible for most cases of anæmia in India. These are firstly a defective diet, which is unable to supply the blood forming tissues in the bone-marrow with sufficient quantities of the chemicals essential for the formation of red blood corpuscles. Secondly there is an increased physiological demand for blood and blood forming substances, as in pregnancy and lactation. Thirdly there is an increased destruction of red cells in the blood and bone-marrow as occurs in chronic malarial infections. Fourthly there is an excessive loss of blood from the body, due to the action of hookworms, the blood-sucking parasites of the intestinal canal. There are, of course, numerous other disease processes, which may be responsible for causing anæmia, but these four factors are far and away the most common. They often operate in combination and not seldom all four may be co-operating together in a single case.

PHYSIOLOGICAL CONSIDERATIONS

Many factors control the development of the red blood corpuscles from their primitive precursors. Two are especially important for a study of anæmia in relation to diet. These are first iron and secondly a specific hæmatopoietic factor (or factors). The latter is formed from precursors, which are widely distributed in foods, but especially in foodstuffs of animal origin and yeast, by the action of normal gastric juice. After formation it is stored in large amounts in the liver. The nature of the deficiencies of iron and specific hæmopoietic factor will now be considered separately.

Iron. This is an essential constituent of the hæmoglobin molecule. Unless an adequate supply of the mineral is available normal replacements of hæmoglobin cannot be carried out and anæmia develops. In iron-deficiency anæmias there is a relatively much greater reduction in hæmoglobin than in the number of red cells. Each red cell contains less than the normal amount of pigment. It is smaller than normal (microcytic) and paler than normal (hypochromic). This type of microcytic hypochromic anæmia is characteristic of iron deficiency. It arises on diets defective in iron and also when excessive losses of iron from the body occurs, as in repeated small hæmorrhages and ankylostomiasis.

Iron is widely distributed in foodstuffs. It is absorbed largely in the upper part of the small intestine. Recent work has shown that only a very small proportion of the total iron taken in the food is absorbed: the great majority passes through the intestines and is excreted in the fæces. Several factors in the intestine affect iron absorption either favourably or adversely, but the exact circumstances most suitable for absorption are not known. The iron in many foodstuffs exists in a chemical combination from which it cannot be freed and made "available" for use by the body. Sound chemical methods for estimating "available" iron in foodstuffs do not exist. Further the iron content of foods has been shown to vary greatly according to the conditions, under which they are grown. Analyses of the iron content of samples of food taken in different parts of India vary greatly. Accordingly the figures of iron analyses given in appendix 1 and daily iron requirements given in appendix 2 are only the roughest approximations and of very limited value. In practice, it is extremely difficult to state whether a given diet is or is not satisfactory as regards iron. An examination of the blood will, however, readily show whether a patient is defective or not in this mineral. Details of the dosage and type of iron required to remedy iron-deficiency anæmias are given in the section on treatment later in this chapter.

There is some experimental evidence that minute amounts of copper are essential for hæmoglobin formation. This element is usually closely associated with iron and most medicinal preparations of iron contain traces of copper sufficient for physiological needs.

Specific Hæmopoietic Factor. The importance of diet in blood regeneration was first emphasised by the American investigator Whipple in 1920. He and his colleagues made dogs anæmic, through repeated bleedings until their hæmoglobin was reduced to 30 per cent of the normal value. The animals were then put on test diets and the rate of regeneration of hæmoglobin studied. The results are shown in Table 9.

TABLE 9

Hæmoglobin production influenced by diet.

Diet, Grams daily.	Hæmoglobin production (Two-week feeding period). grams.	
Bread 400 	...	3
Milk 450, Bread 400 	...	3
Cream 100, Bread 400 	...	10
Butter 100, Bread 350 	...	15
Asparagus 200, Bread 300 	...	9

Spinach 200, Bread 300	...	...	...	15
Raspberries 200, Bread 300	...	...	...	5
Raisins 200, Bread 300	...	...	...	25
Apricots 200, Bread 300	...	...	...	48
Eggs 150, Bread 300	...	...	...	45
Whole fish 250, Bread 300	...	...	...	13
Beef muscle 250, Bread 300	...	...	...	17
Pig muscle 250, Bread 300	...	...	...	30
Chicken gizzard 250, Bread 200	...	...	...	80
Kidney 250, Bread 300	...	...	...	70
Chicken liver 250, Bread 300	...	...	...	80
Beef liver 300, Bread 300	...	...	...	80
Beef liver 450	...	...	...	95

The extraordinary value of liver and to a less extent of other viscera in the regeneration of hæmoglobin was thus demonstrated. The value of liver in the treatment of anæmias other than those due to hæmorrhage was subsequently shown. Its value has been established both in pernicious anæmia in Europeans and in many cases of macrocytic anæmias occurring in the Tropics. There is now considerable evidence that the specific hæmatopoietic factor in liver is manufactured from precursors in the diet. Beef, rice polishings, wheat germ and yeast have been shown to be rich in these precursors, which can be converted into the specific hæmatopoietic factor by the action of normal gastric juice. The active factor is then stored in the liver. So far investigations have not yet demonstrated the chemical nature of the dietary precursor or of the specific hæmatopoietic factor. Indeed it is not known whether the effects are due to a single substance or to a group of substances. Some evidence suggests that more than one active principle exists. Nevertheless it has been possible to concentrate the specific hæmatopoietic factors in liver and to make potent preparations from liver, containing the active principle, suitable for parenteral injection. Patients can thus be spared the unpleasant necessity of consuming large quantities of liver daily by mouth.

In anæmias due to an insufficiency of liver factor, the principal defect is in the normal maturation of the red cell from its large primitive nucleated precursor. There appears to be little or no defect in hæmoglobin production. Thus each cell is usually fully saturated with hæmoglobin (orthochromic). The red cells are indeed usually larger than normal (macrocytic), but great variations in size occur and also there are often many irregularities in shape. A macrocytic orthochromic type of anæmia is commonly found in India, associated with a poor dietary intake and also often a chronic malarial infection.

EPIDEMIOLOGY

There is no doubt that anæmia is widespread throughout India, especially in places where malaria, ankylostomiasis and malnutrition flourish. Circum-

stances have been such that it has been made a special study in three sections of the population, namely the child-bearing women of the large cities, tea garden labourers in Assam and sepoys, especially those recently engaged in the Burma theatre of war. A brief account of the nature and extent of the anæmias present in these groups will now be given.

Anæmia in Pregnancy. The importance of anæmia in the causation of the heavy maternal mortality in India has already been noted in Chapter 1. Napier (1940) in an extensive review of the literature of the subject emphasises the very high maternal mortality in India, figures varying from 15 to 40 deaths per thousand births, being recorded in different surveys. In many of these surveys anæmia occupies the second highest place, as a cause of death and in some first. Anæmia is often a contributory factor in deaths from other causes, notably puerperal sepsis. The condition is distributed amongst all classes of the population, but the poorer sections are most affected. There is no common etiological factor, the usual causes of anæmia being operative in patients, particularly susceptible to their effects owing to the extra demands on the blood-forming tissues made by the fœtus. In consequence a variety of hæmatological pictures are present. A microcytic hypochromic iron-deficiency anæmia and a macrocytic orthochromic anæmia associated with a lack of, or an inability to use, the liver factor are both frequently found in pregnant women. Although the extra demands of the fœtus may clearly strain to breaking point a limited supply of dietary factors essential for red cell formation, there is some evidence that the effect may be more complex. In many cases the response to energetic dietary and liver treatment is poor until the uterus is empty. Napier and Neal-Edwards (1942) write: "There is evidence in addition of some toxæmia associated with the actual presence of the fœtus, which inhibits hæmopoietic response in severe forms of anæmia when in the later months of pregnancy the deficient substances are administered in full doses, although, after the uterus is emptied, there is an immediate maximum response: the toxæmia is probably a 'conditioned toxæmia' which only operates when there is an associated specific food deficiency, either absolute or relative."

Beside the mother, there is one class of person for whom the supply of iron is particularly important—infants and small children from 6 months to 2 years. Amongst them it has been shown that in England a form of nutritional anæmia due to lack of iron is common. Milk is a poor source of iron and the young baby is supplied with a store in the liver, which normally lasts for 6 months approximately. After this age a dietary source of iron is required. In India, where infants are often kept at the breast for prolonged periods, this type of anæmia may be extremely common and a potent factor in the enormous mortality of the early years. There is room for investigation on this problem in India.

The particular tragedy of anæmia in pregnancy is that the great majority of it could be prevented by adequate medical attention in the early antenatal period. This will be referred to again in the section on treatment. The waste of health and life from this cause is an outstanding piece of evidence of dreadful circumstances of present day social conditions in India and of the inadequacy of medical care.

Anæmia in Tea-Estate Labour. Up till now Tea-Estate Labour has been one of the largest and most important single labour forces in India. For this reason a greater study has been made of their health than that of other comparable labour groups. The tea industry has been at some pains to investigate the causes of ill-health in their employees and several medical surveys have been carried out. In many cases it has been possible to find out the primary causes of the anæmia and, as a result of sanitary or other measures, to reduce greatly its incidence with a corresponding gain in the health and efficiency of the labourers. Anæmia is probably equally prevalent amongst other rural communities. However owing to its insidious nature and less spectacular manifestations it has received far less study and attention from public health authorities than such diseases as cholera, plague, small-pox and malaria. MacDonald (1939) describes one survey involving over nine thousand persons in Assam. The economic importance of anæmia can be gauged from his estimate that out of every 1,000 persons on the estates there was a yearly loss of 3,000 working days and 6 lives. General experience would suggest that these figures are no exaggeration and give a reasonable idea of the wastage of health, life and efficiency that results.

Malaria is a potent cause of anæmia in estate labour. However a single attack of acute malarial fever in an otherwise healthy subject properly treated by chemotherapy does not give rise to anæmia. The healthy bone-marrow can rapidly make good the extra corpuscles destroyed. Repeated attacks and inadequate treatment are necessary to cause a breakdown.

Hookworm infestation is another important cause of anæmia on some estates. This is extremely common amongst all agricultural labourers, who cultivate wet or irrigated lands. For the worms to spread it is necessary for the bare feet to be in contact with water, containing the ova from infected persons and for this reason, it is not so common in industrial cities, the well-booted army and those who cultivate dry land. Heavy infestation produces an anæmia of the microcytic iron-deficiency type. A minor infestation is of little consequence and probably at least 100 worms have to be present to cause anæmia. In heavy infestations causing severe and even fatal anæmia, thousands of worms may be found in the small intestine. Iron is essential to replace this blood loss and, if there is an insufficiency of available iron in the diet, the degree of anæmia may be greatly aggravated; thus it is not always

proportional to the severity of the infestation. Extra iron is an essential part of the treatment of ankylostome anæmia.

Nevertheless many observers, including MacDonald, have considered that malaria and ankylostomiasis are not the whole story of anæmia on tea estates and perhaps not even the most important part of the story. Dietary factors are certainly important. Quantitative surveys amongst tea plantation labourers in an area in Assam, in which anæmia is prevalent, show a typical poor rice-eater's diet, similar to that consumed in rice-eating areas throughout India. This is a point of importance. In the Nilgiri hills in South India there are large groups of tea estates, surveys have shown that the diet of labourers in the Nilgiris is similar to that of labourers in Assam. The diets are compared in Table 10. Their similarity is remarkable.

TABLE 10

Food consumed in ozs. per consumption unit per day.

		Assam.	Nilgiris.
Rice—milled parboiled	...	19.4	18.0
Pulses	...	1.0	1.0
Leafy vegetables	...	0.2	nil
Non-leafy vegetables	...	3.4	3.0
Milk and buttermilk	...	0.5	nil
Vegetable oils	...	0.3	0.4
Meat, fish and eggs	...	0.2	nil

Their defects are obvious. Now severe anæmia of any variety, in pregnant women or other age groups, is not common amongst labourers in the Nilgiris. This statement is based on knowledge of admissions to local hospitals. Hookworm and malaria are not prevalent in the part of the Nilgiris in which the survey was carried out. It thus appears that a poor rice diet contains enough iron, and enough of any other necessary hæmopoietic factor, to prevent anæmia in the absence of malaria and hookworms. Such a diet will supply from 8 to 10 mgm. of iron daily, a quantity probably enough to prevent anæmia in the absence of any special strain on the hæmopoietic mechanisms. The crucial fact is that the Nilgiri diet containing as much iron and other essential blood forming factors as the Assam diet, does not *per se* lead to serious anæmia. It is probable that the poor rice diets so widely consumed in India contain just enough iron for maintaining the hæmoglobin content of the blood at a reasonable level under normal conditions, but that there is no margin of safety to meet increased requirements caused by loss or destruction in disease.

Thus all the evidence shows that anæmia prevention in tea estates demands a combined attack on the problems of prevention of malaria, the improvement of diets and the elimination of hookworms. Isolated campaigns against one of these are likely to be of little value.

Anæmia in the Army. During the War many new recruits came from sections of the population whose normal nutrition was never satisfactory. Amongst these men anæmia was prevalent before enlistment. Both the extent and nature of the anæmia was investigated by an Anæmia Team (Hynes, Ishaq and Morris 1945). Their work was carried out mostly in the north, where a widespread mild anæmia was found amongst troops in base depots.

TABLE 11

(Hynes, Ishaq and Morris, 1945)

Distribution of Hb values (Capillary Blood) amongst North-West Indian Soldiers.

Hb. (g. per 100 ml.)	6	7	8	9	10	11	12	13	14	15	16	
No. of cases	...	1	2	5	13	43	131	256	417	361	113	14
Per cent	...	0.5			1	3	9.5	19	31	27	8	1

If the normal level of hæmoglobin is taken as 14 gms. then two-thirds of the men were below normal. Hæmatocrit readings showed that all the anæmias were hypochromic and most normocytic. In no case was a macrocytic anæmia found. In a second large survey amongst newly joined recruits, just below half of the men had hæmoglobin levels below a standard of 14 gms. Again the great majority of anæmias were hypochromic and normocytic, but five very mild macrocytic anæmia were found. Most of the men, who were anæmic had a hookworm infestation, but the correlation between the severity of the infestation and the degree of anæmia was not close. That the hypochromic anæmia was due to an iron-deficiency was shown by the beneficial effect of iron therapy (See Table 12). This result was repeated in a second

TABLE 12

(Hynes, Ishaq and Martin, 1945)

Average Response of different Hb Classes to 21 days iron therapy.

Hb class.	No. of cases.	Before treatment	After treatment
10	5	9.9	13.4
11	4	11.6	13.4
12	7	12.3	14.2
13	19	13.5	14.2
14	15	14.5	15.1
15	6	15.3	15.8
16	2	16.3	16.2

experiment with controls. Iron was given as ferri sulphas exsiccatus and the daily dose was 20 grains (1.3 gm.). In the second experiment a satisfactory response to iron was obtained with a daily dosage of 6 grains (0.4 gm.). This response to added iron is interesting. The dietary intake was approximately 55.5 mgm. of iron. A normal adequate intake is considered to be less than this and such an amount would be expected to provide a liberal margin of safety. Presumably much of the iron consumed could not be absorbed. The major portion about 40 mgm. was obtained from the atta in the diet. This exists probably for the most part in a form not available to the body. There seems little doubt that a mild iron-deficiency was common amongst recruits in North India and therefore presumably widespread amongst the civil population.

During the first years of the Burma Campaign, many sepoy developed anæmia, whilst on active service, and many cases were evacuated to base hospitals in India. The subject became of such importance that a Central Command Anæmia Conference was held in the end of 1943 and attended by most of the senior army Physicians in India. The Proceedings of the Conference, which were published and can be obtained from the D. D. M. S., Central Command Agra, showed that a great variety of cases occurred. It was generally agreed that the most important causative factors were a defective supply of rations and repeated attacks of malaria. The commonest hæmatological picture was a macrocytic orthochromic anæmia. Hookworm infestations were seldom heavy. The anæmias were often extremely severe and very refractory to treatment. Special anæmia centres had to be set up in India to deal with the problem.

The great majority of cases of anæmia occurring in the Indian Army could be directly attributed to poor hygiene, either in their homes or in the difficult conditions of active service on the Burma border. This was shown by the history of events. In 1944 and before, anæmia presented a major medical and administrative problem. With improvements in rations and well-developed air-supplies, good anti-mosquito measures and discipline, suppressive mepacrine, supplementary feeding of recruits, higher standards of cleanliness in unit lines and the introduction of D. D. T., cases of anæmia in hospitals became fewer and fewer. In 1945 during the reoccupation of Burma under very difficult conditions with much heavy fighting the general hygiene was so much improved that but few cases occurred. In 1946 amongst the well-disciplined and well-cared for troops of the army of occupation in South East Asia anæmia ceased to be a problem of any importance. The Army showed that anæmia in India is a preventable disease. Civil health authorities have yet to profit from this lesson.

HAEMATOLOGICAL FINDINGS

A correct hæmatological diagnosis is essential for rational treatment. The therapy necessary for a macrocytic anæmia associated with a deficiency of

the specific hæmatopoietic factor is entirely different from that required for a microcytic iron-deficiency type of anæmia. Unless the nature of each case of anæmia is determined before treatment is started, blunderbuss therapy can only be used and this will mean in many cases a waste of time and money, spent on medicines and drugs which may be valueless for the particular case. The general practitioner can diagnose correctly the majority of cases of anæmia, if he is provided with a good hæmoglobinometer and a microscope. A reliable hæmoglobinometer is essential for assessing the degree of anæmia and the response to treatment. Clinical judgment in this respect is notoriously difficult. A microscopic examination of a stained blood film, after a little practice, will enable a distinction to be made between the small uniform pale red cells of blood crying out for iron and the large irregular cells, usually well supplied with pigment, which indicate a deficiency of the liver factor. A red cell count is not essential and, unless carried out by skilled and practised hands, it is very inaccurate and may be misleading. A careful examination of a stained blood film will usually give sufficient indication of the right line of treatment required.

If laboratory facilities are available, a complete blood examination should be carried out on a single sample of venous blood in each case of anæmia. This will give a quantitative confirmation of the impression gained from looking at a stained film. The methods described by Napier and Das Gupta in their book "Hæmatological Technique" (Third Edition 1945 published by U. N. Dhur & Sons, Calcutta) are eminently suitable for most cases. There are certain measurements, which can be made on blood, which give valuable quantitative information on the nature of the red blood corpuscle. Firstly, there is the Mean Corpuscular Volume, M.C.V., the mean or average volume of the red cells in a sample of blood. It is expressed in cubic microns and is obtained in the following way:

$$\text{M.C.V.} = \frac{\text{Volume of packed red cells in ml. per 1,000 ml. of blood.}}{\text{Red cells in millions per ml.}}$$

For example, if the corpuscular volume is 40 ml. per 100 ml. of blood and the red cell count is 5,000,000 per ml. then $\text{M.C.V.} = \frac{40 \times 10}{5} = 80.0$ cu micron. The normal value for M.C.V. lies between 80 and 100. The second important figure is the Mean Corpuscular Hæmoglobin Concentration (M.C.H.C.) or the average concentration of hæmoglobin in each red cell, expressed as a percentage of the cell contents thus—

$$\text{M.C.H.C.} = \frac{\text{Hæmoglobin in grams per 100 ml. of blood}}{\text{Volume of packed cells in ml. per 100 ml.}}$$

For example, if the hæmoglobin content is 14 grams per 100 ml. and the corpuscular volume is 40 ml. then

$$\text{M. C. H. C.} \frac{14}{40} \times 100 = 35\%$$

The normal value for M.C.H.C. is 30 to 35%. The importance of these values is that they can provide accurate quantitative measurements of the variations from normal standards. Thus if the M.C.V. value is over 100, it means that on the average the red cells are larger than normal (macrocytic). This is a definite indication for liver therapy. Similarly if the M.C.H.C. is less than 30% it means that the cells contain less than normal amounts of hæmoglobin. Therefore iron is indicated. In some cases of anæmia the M.C.V. is over 100 and the M.C.H.C. is less than 30. This indicates that two deficiencies exist and both iron and liver are necessary. Further if the M.C.V. is below 100, it is both unnecessary and wasteful to give liver and, if the M.C.H.C. is 30 or over iron is not required. These measurements, which can be simply carried out in the laboratory provide concise indication for treatment. However, it must be emphasised that they only confirm impressions from microscopic observations of films, which can be made without laboratory assistance.

It is possible to examine the histology of the bone-marrow directly by making smears of material aspirated from puncture of the sternum. The interpretation of bone-marrow smears is at present a specialist's job and, except in the cases of Kala-azar, has proved of little value in practical diagnosis. However, it is undoubtedly an important technique for research investigations. The interested reader can find full details of both the methods of sternal puncture and their interpretation in Napier and Das Gupta's monograph, which has already been referred to.

CLINICAL PICTURE

The clinical features of anæmia are not striking. The onset is often insidious and progressive weakness may be the only complaint. Signs of an associated nutritional failure (see Chapter 2) may be present, but in some cases of severe anæmia the general nutrition is well maintained. The patients may be surprisingly well adjusted to a low hæmoglobin level and it may be impossible to keep those with hæmoglobin levels below 3 and 4 gm. in bed. Hæmic murmurs are usually heard in severe cases and there may be clinical evidence of cardiac dilatation. Yet sudden heart failure is unusual. In fatal cases, the end is usually gradual with progressive weakness and terminal cardiac failure and œdema. Subcutaneous hæmorrhages and severe uncontrollable oral hæmorrhages are common terminal events.

Fever is almost universal in severe cases. It is independent of malaria or other infections. Characteristically it is quite irregular, but occasionally a sustained temperature chart resembling enteric fever is seen.

Test meals give irregular result and are of little help in diagnosis. Hypochlorhydria is not uncommon, but is not a feature of any one type of anæmia. Associated spinal cord lesions are rare and subacute combined degeneration of the cord is undoubtedly extremely uncommon in Indians.

TREATMENT

(a) *Curative.* The general and dietetic treatment of severe anæmia and malnutrition are very similar. The lines of general treatment indicated in Chapter 2 must be followed. Specific hæmatological remedies can be of little value in the absence of an adequate diet and with uncontrolled infections.

Numerous preparations of iron have been used in the treatment of anæmias. Ferrous sulphate, as a pill, 5 grains (330 mgm.) twice a day is an effective dose. The scale preparation is also effective: it is easier to take, but much more expensive. A suitable prescription is

Ferri et ammonium citrate	gr. 30	}	1.9 gm.
Chloroform water to	one drahm		3.5 ml.

(To be taken three times a day in plenty of water after food.)

The above two preparations are simple and sufficient for all practical purposes. No iron preparations exist, which are suitable for injection. Preparations containing iron in organic form are valueless, as they are not absorbed.

For the treatment of macrocytic anæmias, yeast extracts (marmite, vegemite etc.) and dried yeast have been used as sources of the precursor of the specific hæmatopoietic factor. A daily dose of one ounce of yeast extract or at least half an ounce of dried yeast is essential. Numerous liver extracts are available as a direct source of the specific hæmatopoietic factor. The prescriber must make certain that the brand, which he selects, has been made by a reputable firm and suitably tested for potency. Dosage must be high and sustained. Intramuscular injections of 4 ml. of extract daily for 7 days is recommended. Isolated single injections of liver are valueless. It is repeated and cannot be too strongly stressed that liver therapy is valueless in microcytic anæmias. Many patients have suffered the expense and unnecessary inconvenience of liver injections, when all that they required was a cheap iron preparation. Liver by mouth is a valuable adjuvant and a recipe for a liver soup is given in Appendix 5. Numerous workers in India have reported the value of yeast preparations and liver extracts in the treatment of macrocytic anæmias in the tropics, especially when associated with pregnancy. These remedies are undoubtedly valuable in anæmias, but in the writer's experience based on treating large numbers of severe macrocytic anæmias in the army, they are often ineffective. Many army patients, most of whom had suffered heavily from malaria, proved completely refractory to this treatment. It is probable that a temporary exhaustion of the blood-forming tissues occurs and they are then unable to utilise the specific factors present in liver preparations. These changes occasionally become permanent and a complete aplasia of the bone-marrow develops.

In these refractory cases direct replacement of hæmoglobin by blood transfusion is the only remedy. Army experience showed that large transfusions can be safely given to severely anæmic patients, provided proper precautions are taken and suitable equipment available. This is not the place to consider the technical aspects of blood transfusion, but experience has shown that an efficient transfusion service is essential for the proper treatment of severe cases of anæmia.

(b) *Preventive.* The prevention of anæmia in India is clearly bound up with the major public health problems of the country. The importance of measures for the prevention of malaria and hookworm are obvious. The widespread opening of ante-natal clinics would be a great advance, in that early cases could then be brought under medical supervision before deterioration is marked and at a stage during which treatment is likely to be simpler and most effective. Advice to mothers about dietetics can correct errors, which are attributable to ignorance, but cannot touch the larger problem of the disadvantages of poverty. The routine administration of cheap iron preparation is a measure which may be of value in certain instances, especially in schools and ante-natal clinics. Table 13 shows the effect of giving iron to children in two schools in Coorg, where both malaria and hookworm are rife (Bhave and Bopaiya, 1942). Two tablets (2.5 grains or 0.16 grams) of ferrous sulphate were given daily in each school for 2 months.

TABLE 13
*Hæmoglobin levels before and after the administration
of iron (gm. per cent).*
(*Bhave and Bopaiya*)

		No. of Children.	Enlarged Spleen.	Average Hæmoglobin Initial. Final.	
Mercara School	Group A- Experimental	96	32	10.6	11.2
	Group B. Control	91	28	10.6	10.4
Madapur School	Group A. Experimental	22	18	9.6	10.8
	Group B. Control	23	23	9.7	10.0

The children received the tablets on forty days. The cost of the tablets was Rs. 1-8-0 per thousand. This would mean that one child could get an iron supplement daily for a year for approximately Re. 1/-. The improvement, though not striking, was statistically significant and probably associated with an improvement in general health. We may agree with the conclusions of

Bhave and Bopaiya: "The distribution of iron tablets in an area like Coorg, while it may have some good temporary effect, is essentially a makeshift. What is required is a general effort to raise standards of health and especially campaigns against malaria, hookworm and malnutrition."

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POSTSCRIPT: The value of folic acid as a potent anti-anaemic factor in certain types of macrocytic anaemia is now firmly established. Folic acid is an essential dietary factor for many animals, although some such as the rat and perhaps man can live on supplies elaborately by intestinal bacteria. Folic acid is not itself the haematopoietic factor in liver, but is probably an essential part of the mechanism for its manufacture. What part folic acid can play in both the treatment and prevention of macrocytic anaemia in India can only be determined by future research.

CHAPTER 5

DISEASES PROBABLY DUE TO FOOD POISONS LATHYRISM AND EPIDEMIC DROPSY

LATHYRISM

Definition. A degenerative disease of the spinal cord, giving rise to a paralysis of the limbs, especially the legs; it follows upon a long period of subsistence on diets, in which the principal ingredient is the pulse known as *khesari dhal* (*Lathyrus Sativus*).

History and Etiology. Lathyrism is an ancient disease and in the old Hindu literature, *Bhavaprokasa*, it is written that the "triputa pulse causes a man to become lame and crippled and it irritates the nerves." A vivid account of the disease as it occurred in the Central Provinces over a hundred years ago was given by a soldier, General Sleeman. In his book, "Rambles and Recollections of an Indian Official," published in 1844, the description is so clear and characteristic of the disease as it appears to-day that it may with advantage be quoted in full.

"In 1829 the wheat and other spring crops in Saugor and surrounding villages were destroyed by severe hailstorms and rains and in 1831 they were destroyed by blight. During these three years the "teori" or what in other parts of India is called "Kesari" (*Lathyrus sativus*), a kind of wild vetch which, though not sown itself, is left carelessly to grow among the wheat and other grains and given in the green and dry state to cattle, remained uninjured and thrived with great luxuriance. In 1831 they reaped a rich crop of it from the blighted wheat fields and subsisted upon its grain during that and the following years, giving the stalks and leaves only to their cattle. In 1833 the sad effect of this food began to manifest themselves. The younger part of the population of this and the surrounding villages from the age of thirty downwards began to be deprived of the use of their limbs below the waist by paralytic strokes in all cases sudden, but in some cases more severe than others. About half the youth of this village of both sexes became affected during the year 1833-34 and many of them have lost the use of their lower limbs entirely and are unable to move. The youth of the surrounding villages in which the "teori" from the same causes formed the chief article of diet during the years 1831 to 1832 have suffered to an equal degree. Since the year 1834 no new case has occurred but no person once attacked had been found to recover the the use of limbs affected and my tent was surrounded by great numbers of the young, in different stages of the disease imploring my advice and

assistance under the dreadful visitation. Some of them were very fine young men of good caste and respectable families and all stated that their pains and infirmities were confined entirely to the part below the waist. They described the attack as coming suddenly, often while the person was asleep and without any warning symptoms whatever and stated that a greater portion of young men were attacked than young women. It is the prevailing opinion of the natives throughout the country that both horses and bullocks which have been much fed upon "teori" are liable to lose the use of their limbs, but if the poisonous quantities abound more in grain than the stalk or leaves; a man who eats nothing but the grain must be more liable to suffer from the use of the food than beasts which eat it merely as they eat grass or hay."

Modern investigators have confirmed this account. Thus the disease is always liable to occur following the failure of the rains and a poor wheat crop. The Lathyrus pulse is a hardy plant which can flourish on a limited rainfall, insufficient to support a wheat crop. It is often self-sown. Lathyrism is not likely to develop unless the people have been living for at least six months on a diet, of which the greater part of the bulk is composed of lathyrus sativus. It has been observed that persons, who live on *chappaties* made of equal parts of wheat and lathyrus sativus do not suffer.

The condition may be widespread in times of famine and drought in Central India; the Central Provinces, Bhopal and Rewa States have all been severely affected. Occasional cases and isolated outbreaks have occurred throughout the wheat-eating areas of India. It is doubtful if it exists at all in rice-eating districts. The disease occurs occasionally outside India and is not unknown around the Mediterranean, especially in North Africa and Southern Italy.

Lathyrism is a famine disease and now-a-days affects only the poorest classes. It is often, but by no means always, associated with evidence of malnutrition. It is much commoner in men than in women. Young adults and persons in the prime of life are most frequently attacked.

Although there is little doubt that the disease is closely associated with the ingestion of Lathyrus Sativus, the exact factor in the seeds responsible for the symptoms has not been determined. The disease has never been satisfactorily reproduced in experimental animals. Watery extracts of the seeds contain an amine, which is toxic to ducks and monkeys, but it is doubtful, if this can be responsible for the symptoms. At one time it was thought that the seeds of Lathyrus Sativus were not themselves the cause of the disease, but a weed known as akta (*Vicia Sativa*) the seeds of which are frequently found in samples of Lathyrus Sativus. From these seeds toxic bases were extracted, which on administration to guinea pigs caused signs of paralysis of the nervous

system. However in a recent outbreak in Bhopal, stocks of lathyrus seeds consumed in villages, in which many cases occurred, were carefully searched and no vicia sativa seeds were found. This observation has been confirmed and there can now be no doubt that vicia akta is not the cause of Lathyrism.

Clinical Features. The onset is usually sudden. A patient may go to bed at night well and wake up in the morning paralysed or he may fall down while at work at the plough. In other cases backache and stiffness of the lower limbs are premonitory symptoms preceding by a few days the onset of paralysis.

In mild cases there is a weakness and stiffness of the legs only. In more advanced cases the patient walks with bent knees and on tip toe. The legs are often crossed and a "scissors" gait develops. One or two sticks may be needed for support. In severe cases paraplegia in flexion develops and walking is impossible. The patient pushes himself about supporting his body on the balls of the feet, the buttocks and the hands. This is the stage of the disease, which is probably most familiar to City dwellers in India, since many patients by now completely incapacitated for any agricultural work drag themselves to the larger cities to eke out an existence on the pavement from the charity of passers-by. The paralysis is typically spastic. The knee and ankle jerks are exaggerated with clonus and the plantar response extensor. Muscle wasting is seldom seen. The lesion appears to be strictly circumscribed and limited to the lateral columns of the lumbar cord. Thus sensory changes are not found and the sphincters of the bladder and rectum are usually not involved. The muscles of the abdomen, trunk and upper limbs are not affected. There is no evidence of damage to the brain or brain stem and mental changes and psychological disturbances have not been reported.

No recent pathological studies have been made in India and we appear ignorant of any changes in the cerebrospinal fluid.

Course and Prognosis. After paralysis has set in, recovery is rare. Degeneration is usually arrested after a few weeks and the condition becomes stationary. The general health remains good and patients may live long lives, as hopeless cripples.

Treatment. No specific treatment is of any value. Some of the milder cases may benefit from physiotherapy. The disease, once developed, appears incurable.

Although we are still ignorant of the exact cause, there is no doubt that measures to prevent the consumption of lathyrus sativus would cut down the incidence of the disease. The difficulty is the agricultural problem of finding alternative drought resistant crops. In parts of India, where lathyrism is most common, labourers are paid in kind. They may receive lathyrus seeds, as their

only wage. This is a custom, which it should be possible to prevent. Our knowledge of the disease is sufficient to justify legislation to stop the sale of lathyrus sativus or its issue as wages, unless mixed with an equal part of wheat or other grain. The enforcement of any such order might present difficulties and lathyrism will probably continue in certain areas, as long as these areas remain agriculturally backward. At the present there is no indication of any falling off in incidence and widespread outbreaks are reported for many parts of the country. Fuller details can be obtained from the following papers.

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EPIDEMIC DROPSY

Definition. Epidemic dropsy is a disease characterised by œdema, especially of the lower legs, and heart failure. It occurs in epidemics and is prevalent in Bengal. The disease is caused by the consumption of mustard oil, containing oil from the seeds of the weed *Argemone mexicana*.

History and Etiology. Epidemic dropsy is a disease of Bengal and the neighbouring districts. (See maps 2 and 3). It is rare in Madras and the North of India. The disease has presented a puzzle, which has intrigued the physicians of Calcutta for the last seventy years. The back numbers of the *Indian Medical Gazette* contain accounts of many discussions and arguments concerning the nature of the disease. Four views were widely canvassed, firstly that it was an infectious disease due to a specific micro-organism, secondly that it was a deficiency disease akin to beriberi, thirdly that it was an intoxication due to bad rice, and fourthly that impure mustard oil was responsible.

An epidemiological survey carried out in 1937 by R. B. Lal and the staff of the epidemiological section of the All-India Institute of Hygiene, has given the answer to the problem. In an account of this investigation in which the clues appear as intriguingly as in the best detective stories, it is shown that contagion and bad rice cannot be held responsible. Neither can be closely associated with the spread of the disease. A strong case was made out against mustard oil and in some outbreaks the only environmental factor common to all those involved was a single source of mustard oil. The epidemiological evidence against mustard oil has received confirmation from the experimental production of the disease by volunteers who, after consuming mustard oil used by families suffering from epidemic dropsy, themselves developed œdema. Col. R. N. Chopra and his associates of the Tropical School of Medicine, Calcutta, have confirmed the experimental production of the disease in human beings.

Further work has shown that the toxic factor in the mustard oil is not a part of mustard oil itself, but comes from the oil of the seeds of a plant *Argemone mexicana*. This plant frequently grows wild in mustard fields and the seeds bear a superficial resemblance to the black variety of mustard seeds. Epidemic dropsy has been produced experimentally in man by oil expressed from the seeds of *Argemone mexicana*. The actual toxic component of the oil has not yet been isolated, nor its chemical and pharmacological properties fully determined.

It has now been shown that the epidemiology of epidemic dropsy corresponds closely with the use of mustard oil. The disease only assumes importance in Bengal, Bihar and Assam and in the adjacent district of the United Provinces and Orissa. These are the great mustard oil consuming districts. Small outbreaks have occurred in other parts of India and indeed in all parts of the world. The persons affected have usually been Bengali emigrants, who have been taking mustard oil sent from their homes.

In the endemic area Europeans and Marwaris, who do not normally consume mustard oil, are exceptionally immune from the disease.

Pathology. The exact manner in which the toxin causes its deleterious effects is unknown. The œdema is always associated with a reduction of the serum albumen and specific gravity. The distribution of the œdema is not characteristic of hypoproteinæmia and it is possible that either direct capillary damage or heart failure may be contributory.

Clinical Description. The incubation period is variable, but both in experimental cases and the natural disease it may be as short as three or four days.

The onset of œdema is usually insidious and often preceded by mild diarrhœa, abdominal discomfort and general malaise.

The characteristic œdema occurs on the legs and although it may extend upwards and involve the pelvic regions and hands: the face, trunk and neck are rarely involved.

Cardiac involvement is the rule and this is the most important clinical feature of the disease. Generally it is mild and only manifested by palpitation and breathlessness on exertion. Tachycardia is common. Acute cardiac failure may occur and is always a danger. Associated with it, ascites and enlargement of the liver may occur. It is this cardiac factor, which accounts for the mortality, which is in general low, but may be as high as ten per cent in certain outbreaks.

Other common features are a generalised, but irregular erythematous eruption. This may be associated with itching and burning sensations in the skin. In the majority of cases, there is a low grade irregular fever.

A peculiar tumour of the skin or mucous membrane appears in some cases. These may vary in size from a pinhead to a walnut. They begin as tiny red papules which at first gradually increase and then later dry up, leaving a scar, which may be pigmented. The larger tumours may become pedunculated and secondarily infected. The ugly and unsuitable term "sarcoid" has been used for these tumours, whose exact nature is unknown.

A primary non-inflammatory glaucoma is common. This may lead to serious impairment of vision and even complete blindness.

A secondary anæmia is frequent.

Characteristically, there are no changes in the nervous system and the urine presents no abnormalities.

Diagnosis. To any one familiar with the general clinical features and the epidemiology, the diagnosis should not present difficulties. The only other cause of œdema with which difficulty is likely to arise is beriberi. In the past there has been much confusion between these two diseases, but it is now clear that although œdema and cardiac failure are the principal features of both wet beriberi and epidemic dropsy, these two diseases are quite distinct. In cases of doubt a dietary history should enable the correct diagnosis to be made. A complete absence of any neurological signs should also help to exclude beriberi. If real difficulty persists, the therapeutic response to pure vitamin B₁, injected subcutaneously, should clinch the diagnosis. This will have no effect on a case of epidemic dropsy. Whereas in wet beriberi a diuresis and a relief of the cardiac symptoms will set in after a few hours.

Course. Epidemic dropsy may be an acute or chronic disease, presumably depending upon the period over which the toxic oil has been taken. In severe cases the prognosis depends on the cardiac condition and it is probable that permanent cardiac damage may result. The mortality is variable and usually low, but in some outbreaks it has been as high as ten per cent.

Treatment. No specific treatment is known. Owing to the cardiac involvement absolute rest in bed, followed by a prolonged convalescence, is often necessary. Digitalis and other cardiac stimulants are of limited value. Provided the kidneys are healthy mercury diuretics are valuable.

Knowledge of the disease is now sufficiently great to consider the introduction of legislation to control its spread. The All-India Institute of Hygiene

have shown that the number of stocks of mustard seed in oil mills containing small amounts of argemone seed is so large that their indiscriminate rejection would be impractical. However, a quantitative test has been developed by which estimation can be made giving roughly the percentage of argemone oil in each sample of mustard oil. It is recommended that the sale of mustard oil containing 0.5 per cent or more argemone oil should not be permitted.

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CHAPTER 6

MISCELLANEOUS DISEASES IN WHICH ERRORS IN DIET MAY BE A CONTRIBUTORY FACTOR

TROPICAL ULCER—GOITRE—PEPTIC ULCER—DIABETES—DENTAL DISEASE—CIRRHOSIS OF THE LIVER—URINARY STONE

In this chapter a miscellaneous group of diseases, all of which have been considered at one time to be at least partially attributable to dietetic errors, is discussed. No attempt is made to give a complete description of the clinical features or of methods of treatment. These can be found in general textbooks. The discussion is limited in each instance to an account of the incidence and distribution of these diseases in India in relation to nutrition.

TROPICAL ULCER

This is a chronic ulcer usually located on the front of the lower third of the leg, the ankle or foot. It has been called sloughing phagedæna, Naga sore, Cochin sore and a number of other names.

Tropical ulcer is common in tea estates in Assam and many of the existing accounts of the condition in India have been written by doctors attached to estates in that province. According to Roy (1939), an outbreak in 1926 disabled 35 to 40 per cent of the labour population of tea gardens. Tropical ulcer is also prevalent in coolies in tea and coffee estates in Coorg in South India (Bopaiya and Radhakrishna Rao, 1942). Both Assam and Coorg are "jungly" areas, with a high annual rainfall and a high prevalence of malaria and hookworm. Not much is known about the incidence of tropical ulcer in India as a whole, but enquiries indicate that it is not common in labourers in towns and cities or among settled village populations in developed agricultural areas.

In other countries an association between tropical ulcer and climatic and environmental conditions such as those present in Coorg and Assam has been noted. Tropical ulcer is prevalent in Malaya, Indo-China, Melanesia and many parts of tropical Africa. It has been observed in India and elsewhere that outbreaks tend to coincide with the advent of the rainy season.

The ulcers usually occur in persons debilitated by malaria, hookworm and a defective food intake. There is often an initial history of trauma such as a scratch or prick. Bacteriological examination of the ulcers usually reveals the presence of dark staining bacilli, very fine lightly stained spirochætes (*Treponema vincenti*) and often pyogenic cocci. Claims have been made to have produced ulcers by inoculation with these organisms, but most workers have been unsuccessful.

The epidemiological evidence indicates that, although tropical ulcers are not directly attributable to a poor diet, they rarely develop except on persons in whom the general nutrition has been undermined. Two practical points follow. Firstly in any attempt to stamp out tropical ulcer in a labour force or in any district, attention should be paid to an all-round improvement in the diet of the people. Secondly accepted surgical methods of treatment of ulcers will prove futile, unless accompanied by a general improvement in the nutrition of the patient.

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GOITRE

McCarrison (1917) estimated the number of people in India suffering from goitre as 5 million, and considered that "half-a-million is a low estimate of the number who suffer from its congenital manifestations". Goitre is common in the Himalayas and its foothills and in hilly areas in Assam and Burma. There are areas of endemicity in other parts of India, but the proportion of the population affected in such areas is small in proportion to that observed in endemic areas in Himalayan and Sub-Himalayan India. In some Himalayan villages, McCarrison remarks, the disease is so common that it is difficult to find a man, woman or child not suffering from the disease.

The Kangra Valley in the Northern Punjab is known to be a highly goitrous area. A very high incidence has been observed in certain areas in the United Provinces adjacent to the great wall of the Himalayas. A survey of goitre in this province was carried out by Scott, Bhatia, Lal and Rai (1930), who recorded that in Tirraha, in the Gonda district, 70 per cent of the population was affected.

In India we are concerned mainly with the classical parenchymatous or chronic hypertrophic type, sometimes known as simple goitre, occurring in association with cretinism, deaf-mutism, and other forms of physical and mental deterioration. The classical type is essentially a place disease, usually found in the inhabitants of mountain or sub-mountain tracts far from the sea. In areas of high endemicity livestock as well as the human population are affected. Little or no information is available about the prevalence in India of other forms of goitre—*e.g.* diffuse colloid goitre, lymph-adenoid goitre and childhood goitre, not associated with deaf-mutism and cretinism.

According to McCarrison, the condition is more common in adult life than in childhood and hence its frequency in any given area cannot be assessed by surveys of thyroid swellings in children. He found that in Gilgit its incidence gradually increased with increasing age, reaching its highest peak between the

ages of 40 and 60. The same worker (1927) states that "there is a marked tendency to spontaneous recovery in early cases, following removal from the endemic zone: 'first attacks' rarely proceed beyond a certain point of size, the gland enlarging by a step-like series of seasonal increases; in the endemic zone itself 10 per cent or more of 'first attacks' may recover spontaneously, a figure which varies in different localities". In regions of low endemicity it is said to be more common in females than in males, but where it is highly endemic the incidence in both sexes may be the same. Scott and his colleagues observed that in affected areas in the United Provinces the highest incidence occurred in children aged 12 to 15.

Simple goitre in India is a poverty disease, affecting chiefly cultivators of the poorest class. Even in highly endemic areas, the well-to-do usually escape. Seasonal factors apparently influence the development of the disease; new goitres tend to arise, and old goitres to enlarge, during and just after the rains.

Scott and his colleagues observed a high incidence of deaf-mutism in endemic areas and expressed the view that there is a high correlation between the former and the incidence of thyroid deficiency. Numerous cretins and a general low standard of intelligence are to be found in the worst areas. In such areas "distinct evidence of adult myxœdema may be found in (1) the atrophied skin covering the thick myxœdematous subcutaneous tissues, with sub-clavicular pads; (2) the thick puffy lips and tongue; (3) dull mentality with deficiencies of speech and hearing. Many of the latter may be returned as mental subjects." The same workers recorded that one affected area in the Gonda district was known locally as "the abode of fools", there being a popular belief that human intelligence diminished with length of residence in this area.

An estimate of the prevalence of goitre in India such as that of McCarrison is obviously only a rough approximation. Few systematic surveys have as yet been carried out. It is, however, abundantly clear that goitre is one of the most common and serious nutritional diseases found in certain parts of India and a public health problem of great importance. The few investigations which have been carried out in areas of high endemicity give a picture of a miserable, almost sub-human population, deformed, sluggish and degenerate.

It is not proposed to discuss the etiology of goitre in detail. The classical theory of origin can be summarised as follows: The primary function of the thyroid gland is the production of the essential hormone thyroxine, which contains 65 per cent of iodine. Simple goitre is initially an hypertrophy of the gland which reacts to a diminished supply of iodine by enlargement—*i.e.* attempts to carry out its functions under adverse conditions. There is a general association between simple goitre and iodine deficiency in soil and water. The most convincing evidence that iodine deficiency is the primary

cause of simple goitre has been obtained by public health experiments in which iodine, usually in the form of iodised salt, has been systematically distributed in goitrous areas. In certain regions this has led to the disappearance of goitre, or to a great reduction in its incidence.

McCarrison has emphasised the importance of other factors besides iodine deficiency in the etiology of various forms of thyroid enlargement. Observations in the Himalayas convinced him that contaminated water supply is an etiological factor of importance. There are unquestionably many other causes which have been found to produce thyroid enlargements of different types in experimental animals. McCarrison and Madhava (1932) found that the gland was enlarged in rats fed on deficient diets. Agents which produced thyroid enlargement included a high fat intake, an excess of lime and deficiency of vitamin A in the diet. Deficiency of vitamin C had the same effect on guinea pigs and diets consisting entirely of cabbage on rabbits. The variety of factors which under certain conditions influence the functioning and size of the gland probably accounts for a certain haziness in the relation between iodine deficiency and simple goitre. But as far as India is concerned there is little doubt that the essential problem is that of iodine deficiency. A systematic survey of the incidence of goitre in endemic areas is required, followed by attempts at prevention by the provision of iodised salt. No doubt public health work of this kind would bring to light further problems for investigation. Until something of the kind has been initiated, we are in the dark as to the precise extent of the problem and the feasibility of preventive measures. The pioneer work of McCarrison in this field in India has not yet led to practical results.

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PEPTIC ULCER

This is common in certain parts of India. There is little doubt that far more cases are treated in the South than in the North. McCarrison (1921) estimated that peptic ulcer is 58 times more common in the South and Somervell and Orr (1936) that it was 600 times more common. These figures were based on hospital statistics. Dogra (1940) has recently re-examined the question by the study of hospital records and arrives at a ratio of 15:1, representing the difference between Madras on the one hand and the Punjab and the North West Frontier Province on the other. He finds that in the

mainly rice-eating provinces of Bihar, Bengal and Orissa the incidence falls between that of Madras and the Punjab. Too much weight cannot be placed on hospital statistics in a country in which facilities for surgical treatment, skill in diagnosis and operative technique, and the number of surgical operations attempted, vary from centre to centre. But physicians and surgeons in the Government General Hospital, Lahore, rarely see cases of gastric and duodenal ulcer in their wards, whereas in the Government General Hospital, Madras, "gastric cases", the majority of which are cases of duodenal ulcer, constitute 10 per cent of total surgical admissions. Both are excellent hospitals serving large populations. The difference cannot be accounted for on any theory that cases of peptic ulcer occurring in the Punjab are less likely than Madras cases to apply to hospital for treatment, or have their complaint improperly diagnosed and treated on admission. We can therefore take it that the higher incidence of peptic ulcer in the South, first recorded by McCarrison, is a fact and not a statistical fallacy.

Site of the lesion and clinical features. Dogra (1940) made a careful study of 258 consecutive cases of suspected ulcer operated on in the Government General Hospital, Madras, attending each operation himself as an observer. He found the location of ulcers to be as follows:

Duodenum only	...	...	...	...	241
Stomach only	...	...	...	...	5
Duodenum and stomach	...	...	...	...	12

This gives a proportion of gastric to duodenal ulcers of 1 to 48. Somervell (1927), working in Travancore, recorded a proportion of 1 to 32. These ratios may be contrasted with ratios varying between 1 to 1 and 1 to 6 recorded by Surgeons in Great Britain.

Clinically the majority of cases encountered in South India present the classical signs and symptoms of duodenal ulcer. In the early stages there is hunger pain occurring at regular intervals in relation to food and relieved by taking food. The volume and acidity of the gastric juice is increased. Operation usually reveals a small indurated ulcer on the anterior wall of the first part of the duodenum just beyond the pylorus. In the later stages, there are varying degrees of duodenal and pyloric stenosis resulting from cicatrization of the ulcer.

Perforation and serious hæmorrhage are rare. In a series of 2500 ulcers treated by Somervell and Orr, only 4 cases of perforation were seen. They remark that "these exceptional features are consistent with the known and very striking tendency of the Indian abdomen to chronic rather than acute ailments."

Sex and age incidence. All observers are agreed that there is a great disparity in sex incidence. Gastro-duodenal ulcer in India is largely a disease of males. In Dogra's series there were 6 females to 239 male and Orr states that only 5 per cent of his cases are female. Dogra (1940) in a later more extensive study, recorded a case incidence of 1 woman to 18 men. Such differences cannot be accounted for by differences in the numbers of males and females admitted to hospital. In Europe also duodenal ulcer is more common in males than in females.

Social incidence. Dogra remarks that in South India duodenal ulcer "is almost entirely a disease of the poor agriculturist and labouring classes, amongst whom all castes and creeds are affected in proportion to their population. It rarely occurs among the well-to-do." The same worker found it to be very rare among Brahmins, whose economic position is in general higher than that of non-brahmins and the depressed classes. This is a finding of importance, fully substantiated by hospital experience. A similar observation was made by Somervell (1927).

Etiology. Several workers have urged that diet deficiency or error is the chief cause. In support of this theory is the fact that peptic ulcer is uncommon in the north, where the diet is based on wheat and milk and common among South Indians consuming a poor rice diet. The social incidence in South India is also suggestive. Somervell (1927) and Somervell and Orr (loc. cit.), working in Travancore, expressed the view that a tapioca diet, very deficient in vitamins, was an important cause of peptic ulcer. They observed that in Travancore the disease is most common in tapioca-eating districts. Later observations have shown that peptic ulcer occurs all over South India, and there are no grounds for regarding it as a disease specifically associated with a tapioca diet. Somervell and Orr also noted that ulcers were uncommon among fishermen, who consume plenty of fresh fish. Another observations of interest made by them was the rarity of ulcers among State Troops in Travancore, who live in barracks on a well-balanced diet. "In the last five years there have been only two (unproved) cases of duodenal ulcer among them; yet these same men come from villages where there is an incidence of duodenal ulcer of something like one per cent of the population."

Orr and Rao (1939) carried out a careful histo-pathological study of the stomach and duodenum in human cases of peptic ulcers and dogs and rats fed on various kinds of deficient diet. In the human cases they observed pathological changes in the mucosa and the intra-mural nerve plexus of the stomach and duodenum (Auerbach's plexus) at a considerable distance from the peptic ulcer. Very similar changes were noted in the experimental animals. The authors very tentatively suggested that degeneration of the plexus "may lead to spasm, hyper-secretion and abnormality in function of the delicate mechanism

of the pyloric antrum and pylorus" and predisposes to gastric ulcer. The condition of the plexus in the experimental animals was unquestionably due to diet deficiency. This is an interesting idea, worthy of further attention. A view widely held in Europe is that peptic ulceration follows abnormal peristalsis and hypersecretion of gastric juice probably initiated in these cases by psychosomatic disturbances in the hypothalamic region of the brain. The weak point in Orr and Rao's theory is that there is no proof that the nerve lesions which they observed do actually cause functional abnormalities.

Dogra (1940) points out that while the average South Indian labourer or agriculturist may take meals at regular intervals, the interval between meals is often very great and their food is unattractive and monotonous to an extreme degree. "In an average agriculturist family all the adults wake up before sunrise and proceed to work without any food. The first meal, usually taken at about 8 to 9 in the morning is cold and consists of rice cakes or millet left over from the night before along with pickles or raw onions and salt. The next meal is at midday consisting of rice or millet supplemented with "kolambu"; "Kolambu" is a thick mixed stuff made by boiling tamarind, dhals, vegetables, salt, chillies and condiments. Long after sunset, when all the work in the fields, including attending to cattle, is finished and the women have had time to cook food, the last and the principal meal of the day is eaten. This is a hot meal freshly prepared and consists of rice (boiled) with usually vegetable curry. No fresh fruits are eaten. Plantains are an occasional luxury, which are cultivated to be sold. Those that are not sold are dried and utilised for making curries. No milk or milk products form any part of the South Indian agriculturist's diet." The monotonous and unappetising nature of this type of diet, scarcely relieved by the addition of condiments, and the habit of distending the stomach once a day with rice, may play a part in the production of peptic ulcer in India.

But this is mere speculation against any theory that the disease is due primarily and solely to dietary causes in the relative immunity of women, who eat the same food at the same intervals as the men. Dogra does not consider excessive smoking and indulgence in alcohol, habits to which males are more prone of great etiological significance. The disease is a serious problem, both because of its high prevalence and the amount of individual suffering it causes, and requires further careful investigation. If doctors in the East have as yet no solution to offer, they may console themselves with the reflection that the etiology of peptic ulcer in Western countries is still quite obscure.

Treatment. The principles of treatment followed in European countries apply equally in India. Most acute ulcers will heal following a period of complete rest in bed on a diet limited to small, but frequent milk feeds. Owing to the chronicity of many ulcers and the frequency of obstructive

symptoms, many cases come to the surgeons. Gastro-enterostomy is a common operation in South India, popular with both surgeon and patient.

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DIABETES

A complete account of the dietary treatment of diabetes mellitus is not within the scope of this book and the reader is referred to the standard textbooks of medicine. However a brief account of certain aspects of this disease, which is of considerable importance amongst a section of the population in India must be given.

The modern tendency is to divide diabetes into two main types. In the first the patients are usually adolescent or below middle age: loss of weight is often great: ketosis is marked and there is a considerable danger of coma: the patients are usually sensitive to insulin, but even with a careful dietary require large doses, which must be maintained for the duration of life. The prognosis is correspondingly uncertain. In the second type the patients are usually over forty years of age and overweight, even markedly obese. There is often an associated arteriosclerosis and a corresponding liability to gangrene as a complication. Ketosis is not marked and coma uncommon: these patients are sometimes relatively insensitive to insulin, but they frequently respond well to dietary treatment alone and may even be able to dispense with insulin altogether. The prognosis is correspondingly good with due respect to their age. Although this division is by no means exact and there are numerous cases, in which the clinical features do not fall into either type, it is a useful conception and is of some practical value.

There is no statistical data about the incidence of diabetes in India. The general impression is that cases of the juvenile type are probably as common as in other parts of the world. These cases may occur amongst all classes of society, and the majority are found amongst the poor and illiterate. Treat-

ment must be carried out along standard lines and the continual use of insulin is often imperative. The poverty of these patients and their lack of education and corresponding knowledge of the nature of their complaint makes the prognosis very poor in many cases.

There is no question that mild senile diabetes associated with obesity is exceptionally common among the middle and upper classes in India. Indeed it has been said that a mild glycosuria is a sure sign of a successful business career. There is little doubt about the etiology of this condition. Over-feeding and under-exercising are the potent factors. There is no evidence that excess of any particular food substance is responsible. The condition occurs amongst both rice-eaters and wheat-eaters, vegetarians and meat-eaters and is not necessarily associated with any excessive consumption of fat. Sport and exercise, which in the West so often enable successful men to compensate in part on Saturday afternoons and Sundays for excesses during the week at the table, is not a feature in the lives of many of their Indian counterparts.

This type of patient often responds extremely well to treatment, if it is conscientiously carried out, and is often able to dispense with the use of insulin. The essential part of the dietary treatment is a restriction of the calorie intake. It seems probable that experience will show that a "balanced" diet will usually be the most satisfactory and that the artificial high fat and high carbohydrate diet, which may be so irksome and difficult for the patient will be restricted to special cases only. There is no doubt that a diabetic is best upon the minimum amount of food which will enable him to do his work. The drawing up of the details of a dietary regime can only be done by a physician after a full study of the clinical and biochemical features of each case. It is hoped that the food tables published in the Appendix will be of use to Indians suffering from diabetes. It must be emphasised that these values are only approximate and in certain cases may vary considerably. Too high a degree of accuracy must not be sought for in the planning of a diet. The patient's life must not be made a burden.

DENTAL DISEASE

Both dental caries and periodical sepsis are prevalent in India. Despite a wealth of observation and experiment we are still strikingly ignorant of the real cause of these almost universal complaints and correspondingly are unable to prevent their occurrence. In the last ten years several surveys have been undertaken to find out the incidence of these conditions in India and their association with nutrition. These surveys have been undertaken by Dr. Marshal Day of the de Montmorency College of Dentistry, Lahore and Dr. K. L. Shourie of the Coonoor Laboratories. All the figures quoted below are from their papers.

Shourie examined 6866 children in various part of India. The percentage free from caries was 44.5, which may be compared with figures from other countries:

	Per cent free from caries.			
London	...	...	...	1.9
Sheffield	...	...	...	2.5
Lewis, Scotland, rural	...	...	...	28.1
Lewis, Scotland, urban	...	...	...	2.1
Great Britain (10000 English and Scottish children)	...	...	...	14
United States	...	...	...	3.15
New Zealand and Australia	...	...	...	3.5
India	...	...	...	44.5

It was noted that pronounced degrees of dental caries calling urgently for treatment were uncommon in Indian children. His investigation established the fact that dental caries is much less common in India than in the countries of Western civilisation.

Rice-eating children showed slightly more caries than wheat-eating children and the incidence was higher in urban than in rural children in all age groups. A caries incidence of 94.0 per cent was observed in relatively well-to-do urban children in Lahore, while in rural children in the Punjab 41.9 per cent were caries free. But while in the urban group the percentage of children with caries was high, detailed analysis of the data revealed that the percentage of *teeth actually carious* was much lower than in American children.

The consumption of rice diets poor in most of the important vitamins including vitamin D and also in calcium, and generally very deficient by European standards, is compatible with *relative* freedom from dental caries. The diets of Indian children, whatever the staple cereal, contain few "hard" foods which require vigorous chewing. Rice diets are particularly soft and "pappy". Investigations in India thus give no support to the once popular theory that dental decay is caused by the excessive consumption of soft "over-cooked" foods. They also put out of court the theory that vitamin C deficiency is concerned. The best teeth were actually observed in a group of children in Delhi Province consuming small or negligible amounts of fruits and vegetables.

The possible role of vitamin D deficiency in causing dental decay has been the subject of much discussion and investigation during recent years. The generally low incidence of dental caries in India may be compatible with the vitamin D deficiency hypothesis, since in countries bordering on, or in, the tropics more vitamin D is supplied by the action of sunlight on the skin than

in temperate zones. However on the other hand it is well-known that rickets and osteomalacia are more common in North than in South India. Yet the incidence of caries is less in the Punjab than in Madras and the incidence of caries has been shown to be low in children with clinical evidence of rickets in the Northern Punjab.

One observation of interest may be recorded. The highest incidence of caries observed in India was in an Anglo-Indian Orphanage in the Nilgiri Hills near Coonoor. The percentage of children free from caries in the orphanage was only 17.6, and the "average caries figure" was three times the general average for all Indian children. Judged by the usual standards, the diet in the institution was good. In certain respects it was superior to that of poor wheat-eating Indian children, and in nearly every respect superior to that of poor rice-eating children. The allowance of milk products was the liberal one of one pint of whole milk and $\frac{1}{2}$ oz. of butter per child daily. The daily diet also included about 4 oz. of potatoes and about 12 oz. of other vegetables and fruits. The vegetable ration included a fair proportion of green leafy vegetables. Meat intake was about 2 oz., possibly a little less. Consumption of sugar was not less than 2 oz. per child daily, which included molasses given at supper time to spread on the bread. The children also had the opportunity of buying sweets in the school shop once a week up to the amount of their weekly pocket money of 4 annas. The amount of sugar so obtained cannot be estimated; probably in the case of certain children fond of sweets, it was not inconsiderable.

The diet would be regarded as reasonably satisfactory in any children's charitable institution in England. The general health of the children was good and their growth rate satisfactory. They were accustomed to spend plenty of time in the open air, with bare arms and legs, so that there seems to be little possibility of lack of vitamin D. Attention may, however, be drawn to the high intake of sugar, which was greater than that of Indian children in general, and to the considerable quantities of white bread consumed. We know that in countries in which the incidence of dental caries is high, these two foods bulk largely in the diet.,

Whereas Indians, compared with people from other countries, are relatively free from dental caries, they are extremely prone to periodontal disease. Hypertrophic gingivitis is especially common in children, eighty per cent of a group in Lahore being affected. Severe pyorrhœa, often with active pus formation, is extremely common in the middle aged throughout India. Neither of these conditions are closely associated with a poor food intake or with any evidence of malnutrition. There is a large European and American literature describing laboratory experiments and clinical experience, which suggest that these conditions may be attributable to vitamin deficiencies, especially vitamin

C and nicotinic acid. Yet in India a controlled experiment in Lahore, showed that large doses of vitamin C had no effect in the incidence of gingivitis in children. Extensive pyorrhœa is frequent in the absence of any signs of nicotinic acid deficiency and only occasionally does definite improvement follow upon nicotinic acid therapy.

To sum up, dental caries is less common and dental infection more common in India than in Europe. No convincing reasons for these differences are available. The matter remains a fine subject for speculation and future research.

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CIRRHOSIS OF THE LIVER

Cirrhosis of the liver is certainly commoner in India than in Europe, but accurate figures of its incidence in the country are not available: one or more cases of the condition, usually in an advanced state with ascites, is an only too familiar feature of the medical wards in many hospitals. No epidemiological data based on a large series of figures is available, but the general recorded impressions of a large number of physicians is such as to suggest that it is distributed throughout all sections of the population. One particular type of cirrhosis that has been widely studied is that which is not uncommon in children between the ages of six months and three years and which is known as "Infantile Biliary Cirrhosis."

Much has been written concerning the causation of cirrhosis of the liver in the Indian Medical Press during the last sixty years but opinions are for the most part speculative and based on little epidemiological or experimental data. An unsuitable diet has frequently been blamed and hypothetical dietary toxins and deficiencies postulated. Recently there has been a great increase in interest in diseases of the liver throughout the medical world. Post-mortem and liver-biopsy evidence, collected mostly in Africa, has shown that fatty degeneration of the liver is one of the earliest features of malnutrition and is common to a variety of deficiencies and also to semi-starvation. Hepatic damage can also be produced in experimental animals by a variety of dietary deficiencies, notably by a lack of choline and the amino-acid methionine. In most of the experiments acute fatty degeneration or severe necrosis have been produced, but it is not difficult to imagine that partial deficiencies lasting over several years might lead to a widespread cirrhosis. Clinical and experimental investigations of the condition are likely to be pushed forward with energy

both in India and elsewhere. It is to be hoped and expected that a more exact knowledge of etiology and earlier methods of diagnosis may make available methods of treatment to replace the present therapeutic hopelessness. The reader interested in a further study will find the important references in the following two papers.

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URINARY STONES

These are undoubtedly common in certain parts of India and as usual when a disease of obscure origin occurs amongst a population living on a defective diet, it has often been suggested that a food deficiency is a factor in the causation of the disease. In this particular case a vitamin A deficiency was incriminated. This suggestion received support from the well-established fact that vitamin A deficiency causes a transformation of epithelial cells in all parts of the body into stratified epithelium, which subsequently may undergo extensive keratinisation. Fragments of shed keratinised epithelium might well provide the central nidus of organic matter, which is so frequently present in stones and around which the mineral salts are deposited.

In experiments with animals it is possible to produce urinary calculi in rats fed on a diet totally devoid of vitamin A activity and containing an excess of lime. Animals so fed have developed stones regularly after three or four weeks. However, it must be emphasised that such a diet is entirely artificial and in a large experience of feeding both rats and monkeys on defective diets similar to those consumed in India, deficient in, but not totally lacking vitamin A activity, stone formation has never been observed.

The epidemiology of the disease in India does not support the view that it is primarily a deficiency condition. No statistics are available, but stone is always stated to be exceptionally prevalent in Sind, the Punjab and the North West Frontier Province. Certainly it is not exceptionally common in Madras and operations for lithotomy appear to be no more common there than in say London. Yet in Madras there is a widespread action of the population living on diets defective in vitamin A activity and numerous cases of disease directly attributable to this deficiency are found. By contrast to the people of Madras, the people of the Punjab consume far more whole milk and ghee and most of them have an intake of vitamin A up to a reasonable standard. The diets of Sind and the North West Frontier Province resemble those in the Punjab and these three provinces are possibly the best fed in India. Thus there is little support for the view that the prevalence of stone amongst them can be attributable to faulty food. They have, however, the distinction of being the

hottest parts of India during the summer and also the driest. Consequently in these parts in the summer, perspiration must be at a maximum and urinary secretion at a minimum. Hence there must be both an increased concentration of salts in the urine and a diminished flow of fluid which washes out any small deposits. It is suggested that the increased incidence of stone in these three provinces is of climatic rather than dietetic origin.

Nutrition workers are sometimes consulted by persons liable to stone on the nature of the diet best suited to prevent a recurrence of their complaint: on one occasion a patient presented a long list of forbidden articles of diet, which had been made out by a distinguished urological surgeon. The list was clearly composed with the object of reducing to a minimum the production of uric acid in the body and the intake of oxalic acid. Strict compliance with this regime would have made the patient a confirmed valetudinarian and greatly reduced the pleasures of the table. In the present state of our ignorance of the real causes of urinary calculi, such restrictions are unjustifiable. There is no reliable evidence available that increased ingestion of purines or oxalic acid predisposes to stone formation. The only sound prophylactic measure would appear to be the ensurance of a liberal intake (four to five pints) of water daily and this measure must be doubly important in a hot dry climate.

APPENDIX 1

TABLES OF FOOD ANALYSES

A thorough chemical analysis of most Indian foods has been carried out. Two sets of tables have been published and figures for the composition of most foods can be found in "The Nutritive Value of Indian Foods and the Planning of Satisfactory Diets", Health Bulletin No. 23, written by the staff of the Coonoor Laboratories and obtainable through Government Publication agents, price two annas, or in "Field Service Hygiene Notes India 1945" an army publication, which civilian doctors will find a storehouse of accurate and useful information. These tables are very comprehensive, but in practice often rather complicated. The purpose of the tables may be described as follows:

(a) To calculate the composition of diets from data obtained in surveys and from the schedules of schools and institutions. The results can then be compared with standards (see Appendix 2) which state requirements in terms of calories, protein and other necessary food factors. A similar method is followed in devising diet schedules *de novo*.

(b) In order to improve diets, *i.e.*, to make them conform more closely to standards, knowledge of the composition of foods is necessary so that the most effective supplements may be chosen. This will apply equally whether the task in hand is the improvement of the diet of a province with a population of millions by a re-orientation of agricultural policy, or the improvement of the diet of a school, feeding 50 children.

(c) To prevent or cure some deficiency disease associated with the lack of some particular food factor, it is desirable to know how the factor is distributed in foods.

For most of these purposes rough and ready estimates are all that is required. A knowledge of the composition of the different classes of foodstuffs consumed is usually sufficient and analytical details of each individual foodstuff are often not required. Human diets are composed of cereals, pulses, vegetables, fruits, flesh food, etc. Cereals as a group may vary to a certain extent amongst each other, but the group has nutritive properties in common, which differ from those of other groups. The same can be said of pulses, leafy vegetables, or any of the other principal groups of foodstuffs.

In the Tables which follow an attempt is made to give a simplified picture of the nutritive value of the different groups of foods. This is not an easy task because in each group there are exceptions which do not quite fit into the scheme. A range is given and a "selected value" for each chemical component. This value is selected from a knowledge of the variation of the composition of different members of the group and of the variation of different samples. This

method of presentation is open to criticism. Differences in composition between foods belonging to the same group may at times be very important and the simplified tables do not bring out fine distinctions. Nevertheless it is convenient, in dealing with practical nutrition problems, to consider each group as a whole and carry in one's mind a composite picture of the properties of the foods which compose it. In practice it has been found that this method of approach is very useful.

The figures do not make allowances for the losses on cooking, which vary according to local custom. The sources of loss are in the main destruction by heat and extraction into washing or cooking water, which is thrown away. It is not possible to lay down simple factors by which corrections can be made for these losses. Table 14 is a rough and ready guide taken from "Field Hygiene Notes, India."

TABLE 14
Effects of Cooking.

Vitamin.	Food.	Deduct from uncooked value (%)
Vitamin A	Ghee used for frying	90
	Other cooked foods	10
Vitamin B ₁	Parboiled rice (cooking water not consumed) . . .	30
	Parboiled rice (cooking water consumed) . . .	10
	Other rices (cooking water not consumed) . . .	70
	Other rices (cooking water consumed) . . .	60
	Other cooked foods	25
Riboflavin & nicotinic acid	Rice	As for vitamin B ₁
	Other cooked foods	10
Vitamin C	All cooked foods	50

Losses of mineral salts are not included as these are relatively unimportant. Ordinary methods of preparation and cooking are assumed. The superiority of parboiled rice over other rices is clear from the above data. The large loss of vitamin A from ghee used in frying is also noteworthy.

The vitamin A activity of vegetarian foods is due to the presence of the pigment carotene, which is the precursor of the vitamin. The efficiency of the conversion process in the body and of the absorption and assimilation of carotene from plant foods is very variable. Vitamin A activity is expressed in International Units. One I. U. is the vitamin A activity of 0.6 microgram of pure beta carotene. In order to evaluate the true vitamin A activity of

vegetable foods in the human body, the number of I. U. obtained by calculation from the carotene content should be divided by 3 to make allowances for the inefficient utilisation. The figures of vitamin A activity in the tables are expressed in International Units and corrected in the above manner.

The tables are preceded by brief notes on the nutritive value of each class of food. These should be read carefully. The general practitioner will find that from a study of these notes and the simplified tables he will be able to find the answer to the great majority of dietetic problems that may arise. Only on rare occasions should it be necessary to seek the assistance of a "nutrition expert."

The cereals. These include rice, wheat, the various millets, maize, barley, oats and rye. Rye is unknown in India and oats are consumed only by the well-to-do as a breakfast cereal. Barley and maize are of some importance in certain parts of the country, both being consumed "whole". Most Indian diets are based on rice, wheat or one of the millets, or a mixture of these, the cereal in the poor man's diet supplying about 80 per cent of total calories. The cereals as a class are relatively concentrated foods with a high calorie value per unit of weight. They are all rich in carbohydrate and are often described as "energyproducing" foods. Little fat is present in cereals, whether whole or milled. Their total protein content ranges from 6 to 12 grammes per cent, wheat standing the highest and rice the lowest. Some strains of wheat may have a protein content as high as 15 per cent.

Cereals are fairly rich in phosphorus, but low in calcium, so that the calcium/phosphorus ratio is low. Rice has a low calcium content, a matter of considerable importance in practical dietetics, whereas ragi contains much more calcium than any other cereal. As a group the cereals are not rich in iron. They are poor sources of carotene, and, unless germinated, contain no vitamin C. Whole cereals are a good source of vitamin B₁ and a fair source of nicotinic acid; maize stands the lowest among whole cereals in its content of the latter. Cereals are not rich in riboflavin.

In considering the cereals as a group it is important to bear in mind the effect of milling on nutritive value, and that of washing and cooking in the case of rice. In respect of protein, fat, carbohydrate, carotene and mineral content, the difference between a whole or under-milled cereal and the same cereal in the highly-milled state is probably not very important for practical purposes. But it is highly important as regards vitamin B₁ and other members of the B group. The differences between whole wheat and refined wheat flour and between under-milled and highly-milled raw rice are more significant from the standpoint of nutrition than the differences between whole cereals themselves. This question has been dealt with in the chapter on beriberi.

Milled parboiled rice has to a considerable extent the nutritive properties of a whole cereal. (See Table 15).

The pulses. Intake of pulses in India is usually from 1-3 ozs. daily. Like cereals, pulses have certain characteristics which are common to the group. They have a high protein content and for this reason are an important ingredient in diets composed largely of cereals. They contain more calcium and iron than cereals. Little or no carotene is present. Germinated pulses are a useful source of vitamin C and have often been used as an antiscorbutic when other sources of the vitamin were unavailable. The vitamin B₁ content of pulses is high. Their "anti-beriberi" value has long been recognised. They are not rich in nicotinic acid, being in fact poorer than most cereals. They are, however, superior to cereals in riboflavin content and this is a point of importance since good sources of riboflavin are very limited. (See Table 16).

The vegetables. Vegetables may be divided into 3 groups, leafy vegetables, root vegetables, and what, for want of a better term, may be called "other vegetables". A variety of vegetables of all kinds is eaten in India. Vegetables have a high water content, which varies from sample to sample of the same species according to freshness, temperature, etc. Consequently the percentage analysis of undried samples may show wide variation.

Leafy vegetables. Fat, carbohydrate, protein, vitamin B₁, nicotinic acid and riboflavin are present in leafy vegetables only in small amounts, and they do not contribute materially to any diet in these respects. They are, however, rich in carotene and contain considerable amounts of vitamin C, calcium and iron. Therein lies their chief importance as a food in India. The cereals which form the great bulk of the diet are deficient in some or all of these factors. In a country in which supplies of milk products are very limited, leafy vegetables are of great value as a source of vitamin A and calcium. (See Table 17).

Root vegetables. Except for their high water content, the root vegetables resemble cereals in composition. On a dry weight basis the two groups of foods give a not dissimilar analysis. The chief solid constituent of root vegetables is carbohydrate; they contain appreciable amounts of protein, essential mineral elements, and vitamin B₁, but little or no fat or carotene. The carrot with its high carotene content is an exception in this group. Most of the root vegetables, unlike the cereals, contain some vitamin C. Tapioca contains less protein and more starch than most other root vegetables. Diets composed mainly of tapioca contain insufficient protein. In Travancore where tapioca has partially replaced rice as a staple food, its low protein content is a matter of considerable public health importance. There is no other part of India in which root vegetables are consumed as a major ingredient of the diet. (See Table 18).

Other vegetables. The greater part of the vegetables consumed in India fall into this category. The commonest are brinjal and the various gourds. In general vegetables of this kind contain little protein, fat, or carbohydrate. They are not as rich in vitamins and minerals as green leafy vegetables and they contribute fewer calories than root vegetables. They contain little vitamin B₁, nicotinic acid and riboflavin. Their chief value is a source of vitamin C and (to a lesser extent) of carotene. (See Table 19).

Fruits. Which are largely water, contain energy-yielding substances in small amounts. They resemble "other vegetables" in composition, but occupy a somewhat superior position as regards content of minerals, carotene and vitamin C. The amount of carotene and vitamin C present in different fruits varies very widely. The carotene content of fruits tends to increase as they ripen. Fruits are poor in vitamins of the B group. (See Table 20).

Nuts and oil seeds are rich in protein and fat and of high calorific value. Edible nuts are among the most concentrated of foods. They are fairly rich in calcium, phosphorus and vitamin B₁. Ground-nuts are exceptionally rich in nicotinic acid and further investigation may show that other nuts and oil seeds also have a high content of this factor. The riboflavin content of ground-nuts is fairly high. This group of foods is in general devoid of carotene, the only exception being the fruit of the palm *Elæis guineensis*, from which red palm oil, very rich in carotene, is obtained. Red palm oil is not consumed in India, except in very small quantities. (See Table 21).

Taken in small amounts nuts are of value in making good the defects of typical Indian diets. In normal times ground-nuts are cheap and plentiful and some nutrition workers have enthusiastically advocated their increased consumption, on the grounds that they will supply additional protein and fat. A daily intake of 1-2 ozs. of ground-nuts is to be recommended on the score that it will provide additional protein, fat, vitamin B₁ and nicotinic acid. Ground-nuts tend, however, to be nauseating when taken in large amounts. The idea that poor Indian diets could be "balanced" by the substitution of a considerable proportion of cereal by ground-nuts cannot be supported.

Meat, fish and eggs. Muscle meat is a good source of first class protein. Its fat content is variable; in general that of Indian meat is low. It contains some vitamin A and C and is a fair source of the B₂ group of vitamins. It is poor in vitamin B₁. Fish resembles meat in food value and the same is roughly true of eggs which are, however, richer than muscle meat in vitamin A, vitamin B₁ and probably in certain constituents of the B₂ group. (See Table 22).

The distinction between glandular organs and muscle meat is of importance. Liver is superior to meat in its content of vitamins and is perhaps the

most nutritious of all foods. It has a high content of riboflavin and nicotinic acid. The liver oil of fish is a familiar source of vitamins A and D and the livers of mammals also normally contain these food factors. The question is often asked in India whether meat (with which may be included fish and eggs) is a *necessary* food, a question with far-reaching social and religious implications. The only satisfactory reply is that while meat is physiologically a valuable food, it is not an essential food. A diet based on whole cereals, containing plenty of milk, vegetables and fruit, but no meat, can be completely satisfactory as regards nutritive value.

Milk and milk products. The importance of these as a food in India lies in the fact that milk is rich in most of the constituents in which rice and other cereals are deficient. This applies to cow's, buffalo's and goat's milk. The differences in composition of these three milks are relatively unimportant. All are good foods. The high content of calcium and relatively high content of riboflavin are of particular significance. There is some evidence that cow's milk in India has usually a somewhat low vitamin A content, which is doubtless due to the scarcity of green fodder rich in carotene in most parts of the country, except at certain seasons. The variations in the composition of milk in India as influenced by breed of cattle, locality, season, etc., have been insufficiently investigated. Little is known about the vitamin D content of India milk and milk products. Milk is a poor source of vitamin C, vitamin B₁ and nicotinic acid. Milk powder has the percentage composition of the fresh milk from which it is manufactured multiplied by 8 or thereabouts. Some vitamin C is destroyed in the process, but fresh milk is not rich in vitamin C. Skimmed milk and skimmed milk powder are equivalent to whole milk and whole milk powder deprived of fat and fat-soluble vitamins. (See Table 23).

The composition of samples of unadulterated milk or ghee as determined in the laboratory is one thing; that of the same food as sold and consumed in India is another. There is not enough milk to go round in India and the demand far exceeds the supply. Adulteration of milk products is so common as to be the rule rather than the exception in many parts of the country and particularly in towns and cities. For this reason the statement that this or that person or group consumes a given quantity of milk is often difficult to interpret; "milk" may mean anything from pure rich milk to a mixture more than half water. Milk is commonly consumed as "butter-milk"—*i.e.*, soured milk from which the fat has been removed by churning to be made into ghee. Undiluted butter-milk, sometimes called curds, has the nutritive value of whole milk minus fat and fat-soluble vitamins. Butter-milk is, however, consumed in the most varying degrees of dilution—*e.g.* water up to 4 times the volume of the original milk may be added to make a cooling drink. In dietary investigations, intake of butter-milk should always be stated in terms of the original milk.

Butter and ghee are composed of milk fat and have a high vitamin A content, as well as being a valuable source of energy. The vitamin A content of ghee is, however, largely lost, when ghee is used for frying. The numerous artificial ghees on the market (hydrogenated vegetable oils) are valuable foods, as an additional source of calories. They are, however, devoid of vitamins, unless these have been added artificially.

MISCELLANEOUS

Sugar, gur and honey. For purposes of practical dietetics, refined sugar and unrefined sugar (gur) may be regarded as almost pure carbohydrate, with an energy value of 350-400 calories per 100 gm. Unrefined sugar may contain some iron and other mineral matter. Honey contains some 40 per cent of glucose. It has a remarkable reputation in India as a food with valuable medicinal properties, which is no doubt due to the fact that it is easily assimilable and highly palatable. It is, however, devoid of vitamins, a regrettable oversight on the part of the bee.

Condiments and spices. A considerable variety of these are eaten in India. In general they contribute but little to the protein, fat, vitamin and mineral content of the diet, both because they are not rich sources of these food-constituents, and also because the amounts consumed are small. Chillies may, however, be of genuine value as a source of vitamin C, a daily intake of about 0.5 oz. of fresh green chillies providing about 15 mgm. of vitamin C. Condiments are of importance in that they stimulate appetite and make a monotonous diet more palatable. In diet surveys it is found that the poorest families, living on a monotonous rice diet deficient in both quantity and quality, invariably manage to obtain some condiments to add to their rice.

Toddy, both sweet and fermented, is of negligible food value. Sweet toddy contains a little sugar, sometimes up to 5 per cent. All toddies contain traces of the B group of vitamin and vitamin C, but never in sufficient quantities to justify their consumption as food.

Yeast. Dried yeast is one of the richest sources of the B vitamins. Typical dried brewer's yeast contains approximately the following per 100 grammes.

Protein	Vitamin B ₁	Riboflavin	Nicotinic acid
gm./100 gm	mgm/100gm	mgm/100gm	mgm/100gm
50	2 to 4	3 to 6	10 to 60

Its content of B vitamins is greatly superior to that of cereals, pulses and other common foods. Hence its value in the treatment of deficiency states and as a general "tonic".

TABLE 15.

The Chemical Composition of Certain Cereals.

Cereals.	RICE.				Wheat whole.	Refined Wheat Flour.	Millets whole.	Maize whole.	Barley.	REMARKS.
	PARBOILED.		RAW.							
	Home-pounded.	Milled.	Home-pounded.	Milled.						
Moisture per cent	13	13	13	13	13	13	13	13	13	
Protein, gm./100 gm.	7	6	7	6	12	11	13	11	11	
Fat, gm./100 gm.	0.5	0.5	0.5	0.5	2.0	1.0	2.0	3.0	1.5	
Carbohydrate, gm./100 gm.	78	78	78	78	78	78	78	78	78	
Calories per 100 gm.	340	340	340	340	340	340	340	340	340	
Calcium, mgm./100 gm.	6	3	5	4	40	20	40	10	30	Ragi 300.
Iron, mgm./100 gm.	2	2	2	2	4	2	5	1	8	
Vitamin, A, I.U./100 gm.	Trace	Trace	Trace	Trace	50	Trace	Trace to 100	Trace to 200	Trace	Value for maize refers to yellow maize. White maize contains only traces. Cereals are a poor source of riboflavin.
Vitamin, C, mgm./100 gm.	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	
Vitamin, B ₁ , mgm./100 gm.	0.25	0.20	0.25	0.10	0.45	0.10	0.30	0.30	0.30	
Nicotinic acid mgm /100 gm.	4.0	3.5	2.4	1.5	4.5	1.0	1.7	1.4	3.0	
Riboflavin, mgm./100 gm.	...	0.08	...	0.03	0.12	0.05	0.10	0.13	...	

TABLE 16.

The Chemical Composition of Pulses.

Bengal gram, black gram, cow gram, field gram, green gram, horse gram, khesari dhal, peas, red gram (dhal arhar).

		Range	Selected Value.	Remarks.
Moisture per cent	...	8-15		
Protein, gm/100gm	...	17-25	20	Soya bean 40 %
Fat, gm/100 gm	...	1-5	2	„ „ 20 %
Carbohydrate, gm/100gm	...	55-60	55	„ „ 20 %
Calorific value per 100 gm	...	320-360	330	
Calcium, mgm/100gm	...	100-200	150	Horse gram 280, cow gram & field gram 70.
Iron, mgm/100gm	...	2-10	8	
Vitamin A, I. U./100 gm	...	20-200	100	
Vitamin C, mgm/100gm			Trace.	Sprouted pulses contain from 10-15 mg/100 gm. of germinated grain.
Vitamin B ₁ , mgm/100 gm	...	0.20-0.50	0.30	
Nicotinic acid, mgm/100gm	...	1-2	1.5	
Riboflavin, mgm/100gm	...	0.15-0.30	0.25	

TABLE 17.

The Chemical Composition of Leafy Vegetables.

Amaranth, cabbage, coriander leaves, drumstick leaves, fenugreek leaves, ipomoea, neem leaves, spinach.

		Range.	Selected Value.	Remarks.
Moisture per cent	...	75-95		
Protein, gm/100 gm	...	2-5	2	
Fat, gm/100 gm	...	0-2	1	
Carbohydrate, gm/100 gm	...	1-12	4	Neem 20%
Calorific value per 100 gm	...	30-100	60	„ 160
Calcium, mgm/100 gm	...	100-500	300	Cabbage and lettuce 25-150
Iron, mgm/100 gm	...	1-25	10	
Vitamin A, I.U./100 gm	...	1000-5000	1800	
Vitamin C, mgm/100 gm	...	20-200	50	
Vitamin B ₁ , mgm/100 gm	...	0.03-0.05	0.04	
Nicotinic acid, mgm/100 gm	...	...	0.3	The figure is for cabbage.
Riboflavin, mgm/100 gm	...	...	0.05	„ „

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TABLE 18.

The Chemical Composition of Root Vegetables and Tubers.

Colocasia, onion, potato, sweet potato, tapioca, elephant yam, ordinary yam, carrots, turnips, etc.

	Range.	Selected Value.	Remarks.
Moisture per cent ...	60-85		
Protein, gm/100 gm...	1-3	1.5	Tapioca 0.7 %
Fat, gm/100 gm ...	Trace.	0	
Carbohydrate, gm/100 gm ...	10-30	25	Tapioca 38 %
Calorific value per 100 gm ...	50-150	100	
Calcium, mgm/100 gm ...	20-100	50	
Iron, mgm/100 gm ...	0.5-2.0	0.8	
Vitamin A, I. U. /100 gm ...	0-100	60	Carrot 1000-2000
Vitamin C, mgm/100 gm ...	5-25	15	
Vitamin B , mgm/100 gm ...	0.05-0.10	0.075	
Nicotinic acid, mgm/100 gm ...	0.4-1.2	0.5	
Riboflavin, mgm/100 gm ...	...	0.05	The figure given is for carrot.

TABLE 19.

The Chemical Composition of Other Vegetables

(not including leafy and root vegetables)

Amaranth stem, ash gourd, bitter gourd, brinjal, colocasia stems, cucumber, ipomoea, knol-khol, ladies' fingers, plantain stem, pumpkin, ridge gourd, snake gourd, sword beans, onions, etc.

	Range.	Selected Value.	Remarks.
Moisture per cent ...	70-90		
Protein, gm/100 gm ...	0.3-3.0	1.5	
Fat, gm/100 gm ...	0-0.5	negligible	
Carbohydrate, gm/100 gm ...	2-10	5	Ladies' fingers 58
Calorific value per 100 gm ...	15-50	30	
Calcium, mgm/100 gm ...	20-100	60	Amaranth stem 250
Iron, mgm/100 gm ...	0.5-4	2	
Vitamin A, I.U./100 gm ...	0-300	150	
Vitamin C, mgm/100 gm ...	5-100	20	
Vitamin B ₁ , mgm/100 gm ...	0-0.10	0.05	
Nicotinic acid, mgm/100 gm ...	...	0.3	
Riboflavin, mgm/100 gm ...	...	0.05	

TABLE 20.

The Chemical Composition of Fruits.

Guava, jack fruit, orange, mango, papayya, pine-apple, plantain, tree tomato, etc.

	Range	Selected Values.	Remarks.
Moisture, per cent	75 - 90		
Protein, gm/100 gm	0.5 - 2	1	
Fat, gm/100 gm	0 - 0.5	0	
Carbohydrate, gm/100 gm	10- 25	15	Plantain 25
Calorific value per 100 gm	30- 80	50	„ 105
Calcium, mgm/100 gm	10- 50	20	Lime 90
Iron, mgm/100 gm	0. 1- 3	1	
Vitamin A, I. U. /100 gm	Trace-2500	150	Mangoes 1000-2500 Papayya 1000 Tomato 150-200 Orange 150-200
Vitamin C, mgm/100 gm	Trace-600	30	
Vitamin B ₁ , mgm/100 gm	Trace-0.10	0.05	
Nicotinic acid, mgm/100 gm		0. 5	
Riboflavin mgm/100 mg		0. 05	

A "Selected value" is scarcely justifiable in the case of vitamin A and vitamin C in fruits, since the variation is very large. For example, ordinary plantains and country pears, familiar fruits in India, contain less than 50 I.U. of vitamin A per 100 gm., whereas certain varieties of mangoes may contain 30 times as much. Similarly, the vitamin C content of amla and country guava is of the order of 600 and 300 mg./100 mg. respectively, whereas apples, bananas, plantains, figs, melons, avocado pears and pomeloes, mangoes and other fruits contain less than 20 mg/100 mg.

TABLE 21.

The Chemical Composition of Nuts and Oil Seeds.

Cashew nut, coconut, gingelly seed, ground nut, linseed, mustard seed.

	Range.	Selected Value.	Remarks.
Moisture, per cent ...	5-8		Coconut 36%
Protein, gm/100 gm ...	18-26	20	„ 4%
Fat, gm/100 gm ...	37-47	40	
Carbohydrate, gm/100 gm ...	19-23	20	Coconut 13%
Calorific value per 100 gm ...	500-600	520	
Calcium, mgm/100 gm ...	50-500	100	
Iron mgm/100 gm ...	1-5	2	Gingelly seeds 10.5 Mustard seeds 17.9
Vitamin A, I.U./100 gm ...	negligible		Oil seeds used for the manufacture of edible oil in India are devoid of carotene.
Vitamin C, mgm/100 gm ...	nil		
Vitamin B , mgm/100 gm ...	0.15-0.35	0.30	
Nicotinic acid, mgm/100 gm ...	...	16	The values given are for groundnut.
Riboflavin, mgm/100 gm ...	...	0.25	„

TABLE 22.

The Chemical Composition of Meat, Fish and Eggs.

	Range.	Selected Value.	Remarks.
Moisture, per cent	70 - 80		
Protein, gm/100 gm	18 - 22	20	Egg 13 %
Fat, gm/100 gm.	4 - 13	10	Very variable
Carbohydrate, gm/100 gm	0 - 3	2	
Calorific value per 100 gm	110 - 190	150	Depending on the percentage of fat present.
Calcium, mgm/100 gm	10 - 70	20	
Iron, mgm/100 gm.	0.7 - 6.0	2	
Vitamin A, I. U./100 gm	Trace -100	40	Liver 10,000—20,000.
Vitamin C, mgm/100 gm	1 - 3	2	Liver and Kidney 10-40.
Vitamin B ₁ , mgm/100 gm	...	0.050	Pork 0.30
Nicotinic acid, mgm/100 gm	3 - 5	4	Liver 15.
Riboflavin, mgm/100 gm	0.05 - 0.30	0.15	Liver 2.5

TABLE 23.
The Chemical Composition of Milk.
 Cow, buffalo, goat.

		Range.	Selected Value.	Remarks.
Moisture, per cent ...	...	80-90		
Protein, gm/100 ml ...	...	3-5	4	
Fat, gm/100 ml. ...	...	3-5	4	Buffalo 9.0 %
Carbohydrate, gm/100 ml	...	4-5	4	
Calorific value per 100 ml.	...	65-85	75	Buffalo 120
Calcium, mg/100 ml	...	120-200	150	
Iron, mg/100 ml ...	...	0.2-0.3	0.2	
Vitamin A, I.U./100 ml.	...	150-200	200	
Vitamin C, mgm/100 ml	...	0.5-2.0	1	
Vitamin B ¹ , mgm/100 ml.	...	0.05-0.10	0.05	
Nicotinic acid, mgm/100 ml.	...		0.1	Cow's milk.
Riboflavin, mgm/100 ml.	...		0.15	„ „

Ghee from cow's milk fat—1000-2500 Int. Units per 100 gm
 Ghee from buffalo milk fat—500-1000 do. do.

APPENDIX 2

(From Report of the Twelfth Meeting of the Nutrition Advisory Committee, I.R.F.A., New Delhi, November 1944.)
Daily Requirements of Calories and Some Essential Nutrients.

		Net calories.	Proteins.	Fats.	Ca (Calcium.)	Fe (Iron.)	Vit. A. I. U.	Thiamin (Vit. B ₁ .)	Vit B ₂ complex.	Ascorbic acid.	Vit. D. I. U.
Man (55 Kg. or 120 lbs.)	Light or sedentary work	2400	82	See note (4) following the table.	1.0	20 to 30	3000 to 4000	1.0 to 2.0	See footnote (10) following table.	50	400 to 800
	Moderate work	3000	82								
	Very hard work	3600	82								
Woman (45 Kg. or 100 lbs.)	Light or sedentary work	2100	67	See note (4) following the table.	1.5 to 2.0	10 to 30	3000 to 4000	0.5 to 1.0	See footnote (10) following table.	30 to 50 and over	400 to 800
	Moderate work	2500	67								
	Very hard work	3000	67								
	Pregnancy	2100	101								
Children	Lactation	2700	112	See note (4) following the table.	1.0 to 1.5	10 to 30	3000 to 4000	0.5 to 1.0	See footnote (10) following table.	30 to 50 and over	400 to 800
	Under 1 year	100/Kg.	3.5/Kg.								
	1 to 3 years	900	3.5/Kg.								
	3 to 5 "	1200	3.5/Kg.								
	5 to 7 "	1400	3.0/Kg.								
Adolescents	7 to 9 "	1700	2.5/Kg.	See note (4) following the table.	1.0 to 1.5	10 to 30	3000 to 4000	0.5 to 1.0	See footnote (10) following table.	30 to 50 and over	400 to 800
	9 to 12 "	2000									
	12 to 15 "	2400									
	15 to 21 "	2400	2.0/Kg.								

N.B.—The estimates of the protein requirements of children and adolescents are given in terms of grammes per kilogram because adequate data about average weight in the various age groups were not available to the Sub-Committee.

NOTES ON TABLE

(1) The term 'net calories' means the energy available from the food actually assimilated.

(2) Additional calories for moderate and heavy work have been provided for, in accordance with the recommendations of the Technical Commission on Nutrition of the League of Nations Health Organization.

(3) Proteins of animal origin are generally superior in biological value to vegetable proteins. It is, therefore, desirable that some animal proteins should be included in the diet. Various estimates have been made of the desirable proportion of animal to vegetable proteins, e.g. 1:1, 0.5:1 or less. These are however not based on a fully satisfactory scientific foundation. Some animal protein should, however be included in the diet.

(4) Fats must be included in a balanced diet but there is no exact knowledge at present available of the quantity required; hence no figures have been included in the table. Fats possesses the advantage of yielding more than twice the energy obtained from carbohydrates or proteins. It is the general experience of nutrition workers that, even in a temperate climate, there is a tendency towards a higher consumption of fats in winter than in summer. A liberal consumption of fats can be advocated on the grounds that some of them act as vehicles for fat soluble vitamins and thus may provide these nutrients to the body in appreciable quantities.

(5) Figures for carbohydrate requirements are not given in the table. If the constituents listed in the table are obtained from a variety of natural food-stuffs adequate amounts of carbohydrate will be obtained.

(6) Equivalents of 1 milligramme of various vitamins in international units are shown below:

1.0 milligramme B-carotene = 1666 I.U. Vitamin A.

1.0 " Vitamin A = 3300 I.U.

1.0 " Calciferol = 40000 I.U. Vitamin D.

(7) Vitamin A requirements can be met by preformed vitamin from animal foods, and by provitamin A (carotene) present in some foods of plant origin. When the latter form the bulk of the source of the vitamin, a higher level of intake is necessary than when preformed vitamin A is the source of supply. In Indian diets provitamin A is the main source of vitamin A activity. The figure given in the table is intended to cover vitamin A requirements in terms of Indian food habits.

(8) Vitamin D is undoubtedly necessary for older children although no definite figure can be given at present. Exposure to the ultraviolet component

of sunlight leads to the formation of vitamin D in the skin and thus may supply a part of vitamin D requirement. No data are available about the contribution to vitamin D requirements from this source in tropical and sub-tropical countries.

(9) The information about the availability of iron from different food-stuffs is incomplete. Hence a figure for total iron intake higher than the usually accepted standard is included in the table.

(10) The human requirements of riboflavin, nicotinic acid and other members of vitamin B₂ complex have not yet been placed on a fully satisfactory basis and hence are not included in the table. These vitamins are, however, essential for human nutrition. A few quantitative estimates of requirements have been made, *e.g.* from 2.2. to 3.3 mgm. of riboflavin and 15 to 23 mgm. of nicotinic acid for adult men. Future research in India and elsewhere should be directed to placing this problem on a firm scientific basis.

(11) There are several other minerals which are essential in nutrition, *e.g.*, iodine, magnesium, copper, manganese, etc. In general, if a diet is well-balanced and is adequate in respect of other better known essential nutrients it can be assumed that it will supply such minerals in adequate quantities.

(12) Allowance has been made for the unavoidability of a certain proportion of most of the constituents in mixtures of foodstuffs, as also for the possibility of destruction through methods of preparation.

APPENDIX 3.

SUMMARY OF THE PROPERTIES OF THE MORE IMPORTANT VITAMINS

It has been repeatedly emphasised in the main text that pure vitamins have a very important, but strictly limited place in modern therapeutics. The number of known vitamins is increasing yearly and with each new discovery, fresh therapeutic claims are made and new preparations are put on the market by chemical manufacturers and widely advertised amongst the medical profession. Many of these cannot live up to the claims of their makers and are doomed to cause expense and disappointment, both to patient and doctor. The following short synopsis is designed to give a concise account of our present knowledge of vitamins, as a background for medical practice and specially therapeutics.

VITAMIN A

Distribution in nature. The primary sources are the plant pigments, known as carotenes. These are red or yellow hydrocarbons, three or four of which can be converted into vitamin A in the body. Carotene is distributed widely throughout the vegetable kingdom, the amount present being very roughly related to the colour of the food. Green leafy vegetables and ripe fruits are rich sources. Other vegetables are unreliable sources, though most contain appreciable amounts and one or two such as carrots are very rich. Cereals contain negligible amounts, except for yellow maize. Vegetable oils are devoid of vitamin A, except red palm oil. (Commercial preparations of vegetable oils may contain vitamin A added by the manufacturers). In the animal body carotene is absorbed from the intestines and converted into vitamin A in the liver. The ability of the body to utilise carotenes in different foods is very variable. On an average only about one-third becomes available as vitamin A. Vitamin A is stored in the liver and a liberal supply is provided for the next generation. Thus liver, liver oils, milk and eggs are good sources of vitamin A. In milk the vitamin A is dissolved in the fats. Hence ghee, butter and cream are very rich sources and skimmed milk contains little or none. Other foods of animal origin contain only small amounts.

Chemical Properties. Vitamin A is an alcohol $C_{20}H_{29}OH$. It is fat-soluble and unsaponifiable. It is resistant to heat in the absence of air, but is destroyed by oxidation at higher temperatures. Losses in ghee and other fats used for frying are very high, up to ninety per cent, but in other methods of cooking losses are small (about ten per cent).

Function and Effects of Absence. Vitamin A is essential for growth. With inadequate amounts children will not grow normally. This vitamin is also concerned with the maintenance of healthy epithelial surfaces. In its absence desquamation of the dead superficial layers of epithelium does not take place and cornification results. In the skin and the eye this leads to the conditions known as nutritional hyperkeratosis, follicular hyperkeratosis and xerophthalmia (see Chapter 2). Further the various epithelial surfaces are not able to function effectively as a first line of defence against bacteria. Consequently vitamin A deficiency leads to an increased susceptibility of infections, especially of the respiratory and urinary tracts and of the skin. Vitamin A is also essential for the regeneration of visual purple in the retina and in its absence night-blindness results. The exact manner in which vitamin A fulfills these several functions is not known.

Requirements and Therapeutic uses. Normal daily requirements are about 3,000 to 4,000 International Units. Children need at least as much as adults. Vitamin A is indicated in all cases of xerophthalmia. It is also indicated for children, who are underweight, cases of follicular hyperkeratosis, chronic infections of the skin and chronic infections of the respiratory and urinary tract, and night-blindness, *provided* a good dietary history has been taken and there is clear evidence that a sufficiency of foods rich in vitamin A are not being consumed. A daily dosage of 10,000—20,000 I.U. units should be given. This amount will be contained in about half to one ounce of a good fish liver oil. Other more concentrated preparations are on the market (see Chapter 2).

VITAMIN B₁

Distribution in nature. Vitamin B₁ is widely distributed in all living tissues. The only important stores in the biological world are in the seed grains in the plant kingdom and in the liver in the animal kingdom. Therefore whole cereals, pulses and liver and in addition yeast are the only rich sources of the vitamin. In cereals the vitamin is present mostly in the outer pericarp and germ and consequently refined highly milled rice and wheat products contain very little. Leafy vegetables, root vegetables, fruits, flesh foods, eggs and milk contain small, but significant amounts of the vitamin, but usually not enough to compensate in diets composed mainly of highly milled cereals for the losses in milling. Refined sugar, fats and oils are devoid of vitamin. Jaggery, gur, toddies, beer and other alcoholic drinks contain negligible amounts.

Chemical Properties. Vitamin B₁ has been synthesised. The pure substance, thiamin, contains a pyrimidine and thiazole ring and has the formula C₁₂H₁₆ON₄S. It is a white crystalline substance, very soluble in water. It

can therefore be easily washed out of foods. In alkaline solution, it is unstable and readily destroyed by heat, but in acid and neutral solution it is fairly stable and losses during ordinary cooking processes are probably about 10 per cent.

Function and Effects of Absence. Vitamin B₁ is an essential factor in the intracellular metabolism of carbohydrates. It functions as a co-enzyme and is part of the enzyme system responsible for the metabolism of pyruvic acid, an essential stage in the metabolism of sugars. In its absence this acid accumulates in the blood and tissues. Nerve tissue seems particularly sensitive to a lack of the vitamin and a peripheral neuritis develops if a diet deficient in the vitamin is consumed for several weeks. This form of neuritis is known as beriberi.

Requirements and Therapeutic uses. On an average diet of about 2600 calories, 0.7 mgm. of vitamin will usually prevent the appearance of beriberi. Any factor which increases metabolism, such as hard physical work or fever, increases requirements. For safety and optimum health a daily intake of 1.0 to 2.0 mgms. is recommended. Pure preparations of the vitamin are available and are indicated in acute cases of beriberi, when they are often life-saving. The vitamin can be given subcutaneously or intravenously. A suitable dosage is 5-10 mgms., which can be given four-hourly, until improvement sets in. Except in acute cardiac beriberi, injections of pure vitamin B₁ are unnecessary. Indeed in chronic cases, mild attacks and for prophylactic purposes, yeast preparations are preferable. There is no evidence that vitamin B₁ injections are of any value in any other forms of chronic neuritis.

B₂ GROUP OF VITAMINS

In the early days of the discovery of vitamins, it was found that the effects of yeast could be attributed to two water soluble factors, which were called vitamin B₁ and vitamin B₂. Vitamin B₁, the antineuritic vitamin, could be readily destroyed by heat, whereas vitamin B₂ which was essential for growth and preventing dermatitis in experimental animals was heat stable. Vitamin B₂ has been shown to comprise a whole group of essential factors. At least eight substances have been isolated in pure form from this group and each shown to possess the properties of a vitamin. These are riboflavin, nicotinic acid, pyridoxine, pantothenic acid, inositol, p-amino-benzoic acid, biotin and folic acid. Of these the first two, riboflavin and nicotinic acid, have been demonstrated to be essential for man and clinical signs and symptoms attributable to lack of these two vitamins have been described. A brief summary of their properties will be given in the next paragraphs. Of the remaining six factors, whose numbers are always liable to increase, it may be stated that all have been satisfactorily demonstrated to be an essential substance for the growth or well-being of a lower organism. There are reasons for believing,

but no satisfactory proof, that each is also essential to man. So far no definite proof exists that any specific signs of disease can be attributed to lack of any of them. However there is evidence which suggests that folic acid is the specific hæmatopoietic factor or it's precursor and that lack of folic acid causes macrocytic anæmias. There is also evidence that the "burning feet syndrome" may be due to pantothenic acid deficiency. Both of these claims need confirmation. Preparations of these substances are already on the market. The busy practitioner can afford to suspend judgment on these factors. Their practical importance in human dietetics is still doubtful: further a good yeast preparation contains all of them and by prescribing yeast, it is possible to ensure a sufficient intake of the whole B₂ group.

RIBOFLAVIN

Distribution. Riboflavin is widely distributed in the plant and animal kingdom, but very few rich sources exist. Whole cereal grains contain moderate amounts, but losses on milling and cooking may be great. Parboiled rice retains more than raw-milled rice, but is not a good source. Vegetables of all kinds contain small amounts only. Fruits are also not rich. Pulses are good sources. Nuts and in particular groundnuts are rich. Milk is a valuable source. Flesh foods contain appreciable amounts. Liver and yeasts are both rich. Evidence of riboflavin deficiency is uncommon in North India. The atta, assisted by a fairly liberal milk consumption, provides a sufficiency. In South India riboflavin deficiency is common. Rice loses much of the vitamin in preparation and milk intake is less. Pulses are a most important source of riboflavin in the poor rice diet.

Chemical Properties. Riboflavin has been isolated from many sources. It is yellow crystalline substance. It is soluble in water and relatively heat stable, although unstable in the presence of alkalis. It fluoresces brightly in ultra-violet light. Riboflavin is composed of an alloxazine ring, to which ribose, a five carbon atom sugar is attached.

Function and Effects of Absence. In the tissues riboflavin forms with phosphates a compound, which is then bound to a specific protein. The resulting flavoprotein is an enzyme, which acts as an oxidase and is essential for the transfer of oxygen in most animal tissues. In man riboflavin deficiency manifests itself as angular stomatitis, genital eczema and superficial keratitis (see Chapter 2) although these conditions are probably not pure riboflavin deficiencies.

Requirements and Therapeutic uses. The minimum daily requirements of riboflavin to prevent signs of deficiency disease are probably 2 to 3 mgm. Pure riboflavin can be given by mouth or subcutaneously in doses up to 10

mgms. and will usually alleviate the symptoms of severe manifestations of deficiency. Minor cases are more cheaply and as effectively treated with yeast or milk.

NICOTINIC ACID

Distribution. This vitamin has a similar distribution to riboflavin and is widely found in most foods. Whole cereal grains are fair sources, but losses on milling may be severe. Pulses and nuts are good sources, groundnut being especially rich. Vegetables, fruits and milk contain small, but variable amounts. Flesh foods are fair sources. Liver and yeast are both very rich.

Chemical Properties. Nicotinic acid, $C_6H_5O_2N$, is a derivative of the base pyridine. It is a white crystalline substance, soluble in water and very stable, not being destroyed by any of the normal cooking processes.

Functions and Effects of Absence. Like riboflavin, nicotinic acid is intimately concerned with intermediary tissue metabolism. It is an essential part of the molecules of two co-enzymes, which are widely distributed in nature and function as hydrogen acceptors in numerous oxidation systems. In man nicotinic acid deficiency is associated with many of the signs of pellagra, especially glossitis and diarrhoea (see Chapter 2.).

Requirements and Therapeutic uses. Daily needs are about 15 to 25 mgms. Pure nicotinic acid can be given by mouth or by subcutaneous injection. A maximum dose is 100 mgm. which can be repeated four-hourly. It is indicated in all severe cases of nutritional glossitis and nutritional diarrhoea. In minor cases and for prophylactic purposes, yeast preparations are more valuable.

VITAMIN C

Distribution. Vitamin C is widely distributed in fruits and vegetables. Green leafy vegetables and ripe fruits are usually very rich. Gourds and similar vegetables are less rich sources. Root vegetables and flesh foods contain small, but appreciable quantities. Milk is a poor source and when boiled contains negligible amounts. Cereal grains and most pulses are devoid of the vitamin. However they form large amounts on germination.

Chemical Properties. Vitamin C has been synthesised. The pure vitamin, ascorbic acid $C_6H_8O_6$, is a white crystalline substance easily soluble in water. It is readily oxidised and is the least stable of all the vitamins: it is slowly destroyed by heat, especially in alkaline solution. Losses due to cooking have been exaggerated and probably about fifty per cent survives ordinary cooking processes.

Functions and Effects of Absence. The essential function of vitamin C is the maintenance of the intercellular ground substances, which support the cells of the tissues. In its absence mesodermal cells fail to develop: capillary hemorrhages result from increased friability and in the young, the formation of bone and dentine is impaired. The healing of soft tissue wounds and fractures is also delayed. Severe deficiency leads to scurvy (*see* Chapter 3).

Requirements. 5 mgm. of the vitamin a day is undoubtedly sufficient to prevent scurvy. Much larger amounts may be needed to maintain satisfactory health and a daily intake of 50 mgms. is recommended. The daily utilisation is increased by fever and infection. Pure ascorbic acid is cheap and readily available for treatment. It is indicated in cases of scurvy and in severe wounds and fractures and in all infections, if the natural dietary intake of the vitamin is insufficient (less than 50 mgm. daily).

VITAMIN D

Distribution. Milk and ghee are the main natural sources. Eggs are also rich. Vegetable foods contain negligible amounts. The vitamin can be synthesised in the human body from precursors in vegetable foods. The synthesis takes place in the skin following irradiation with ultraviolet light or bright natural sunlight. Fish liver oils are a rich additional sources.

Chemical Properties. Pure vitamin D, Calciferol, is a crystalline substance derived from sterols as the result of irradiation. It is soluble in oils and fats and fat solvents, but not in water. It is fairly stable and not readily decomposed.

Functions and Effects of Absence. Vitamin D is concerned with the control of calcium and phosphorus metabolism and the regulation of bone formation. In its absence rickets and osteomalacia develop (*see* Chapter 3).

Requirements and Therapeutic uses. The daily requirements are 400-800 International Units. Vitamin D is indicated in the treatment of rickets and osteomalacia. For therapeutic purposes the daily dose should be 1,000 to 3,000 I. U., which is contained in one-third to one ounce of cod liver oil (B. P. Standard). Concentrated preparations are available and convenient. Care must be taken not to give excessive doses as vitamin D in large amounts is toxic.

VITAMIN E

Function. Rats reared upon a whole milk diet, containing an abundant supply of vitamins A, B group, C and D are usually sterile. In addition a muscular dystrophy, not involving any degeneration of the nervous system,

develops in rats and other animals fed on similar diets. It was found that these effects could be prevented, if wheat germ oil was added to the diet. The action of the wheat germ oil was found to be due to a substance known as vitamin E.

Distribution and Properties. Vitamin E has been isolated in pure form and synthesised. Three crystalline isomeric compounds, each physiologically active, have been isolated. These are alpha, beta and gamma tocopherol and are higher alcohols, soluble in fat solvents and relatively stable. Most vegetable oils contain considerable vitamin E activity.

Clinical Trials. Vitamin E therapy has been extensively tried out on human beings suffering from habitual abortion, various forms of sterility and types of muscular dystrophy. So far all satisfactorily controlled tests have been negative and there is no clinical evidence, that vitamin E deficiency exists in man. Vitamin E preparations are on the market: the practitioner is advised not to use them, unless he has familiarised himself with the literature and the failures of others to produce any benefit with this vitamin and he is prepared to undertake experimental trials.

VITAMIN K

Functions and Effect of Absence. Vitamin K is essential for the normal formation of prothrombin in the liver. In the absence of prothrombin there is a prolongation of the clotting time and a tendency to hæmorrhage. Vitamin K is widely distributed in nature and is present in most vegetables. It is also synthesised by bacteria in the intestinal canal and normal fæces contain large amounts. Deficiency due to an insufficiency in the diet probably does not occur. However deficiency may occur in the newborn, possibly from intestinal sterility, and hæmorrhagic disease results. The vitamin is not absorbed from the intestine in the absence of bile and in adults with obstructive jaundice, hæmorrhages due to vitamin K deficiency may arise.

Chemical Properties. Vitamin K is fat-soluble and unsaponifiable. It is a derivative of naphthoquinone. Several other synthetic naphthoquinones have vitamin K activity and in many cases are more active than the vitamin itself.

Therapeutic uses. A synthetic preparation 2-methyl-1, 4 naphthoquinone is generally used in clinical practice. It is indicated in all hæmorrhagic diseases of the newborn and in hæmorrhage conditions, associated with liver disease. It should be given prophylactically before all operations for obstructive jaundice. The usual dose is 10 mgm. given by subcutaneous injection.

VITAMIN P

It has been claimed that certain vegetable dyes known as flavonals, of which hesperidin is the most important, control vascular permeability. Certain cases of hamorrhagic diseases with increased permeability and fragility of the capillary wall have been reported to have failed to respond to vitamin C, but to have been cured by lemon juice, extracts of red pepper and hesperidin (vitamin P). This work has as yet not received general confirmation and it is uncertain if vitamin P actually exists.

APPENDIX 4
(By Courtesy of the Director of Medical Services, G. H. Q. India.)
Hospital Diets and Extras. Army Instructions (India) 1063/45.

Daily Scale of Diets for Indian troops and enrolled non-combatant personnel.

Issues will be guided by the following scales according to the diet upon which each patient may be placed. The quantities of food for each form of diet plus the extras admissible are the maximum allowance for each diet and must not be exceeded.

Description of diet.	Fluid	Inter-mediate	Light	Low Residue	High Protein	Low Fat	Ordinary	Subsistence	REMARKS.
Reference letter.	F	I	L	R	C	J	O	S	
Cereals etc.									
Atta/rice (a) oz.	...	2	10	10	12	18	20	6	(a) Atta and rice may be demanded in any proportion required, but normally patients should receive a proportion of each. In the case of diet "R", not more than 4 oz. of atta should be issued.
Bread "	...	...	...	4	6	...	...	...	
Biscuit fancy "	3	...	...	2	...	...	...	2	
Cereals for pudding (b) "	...	2	2	1	2	...	...	1	
Dairy produce.									
Milk fresh (lb) (c)	3½	3½	2	3	3	½	1	1	(b) Pudding cereals to consist of arrow-root, sago, tapioca, cornflour, or suji (semolins) when medically essential, particular varieties may be demanded.
Skim milk powder oz.	...	...	...	...	...	6	...	...	(c) Milk tinned may be issued in lieu of milk fresh, equivalent being 4 oz. to every 10 oz. fresh milk.
Cheese "	...	...	...	...	1	...	4	...	(d) To meet the religious scruples of certain classes of patients, meat, fish, eggs, etc., may be substituted on the recommendation of the medical officer in accordance with the following table:—
Eggs (No.) (d)	...	3	2	2	3	...	2	2	Mutton fresh with bone 2 oz. = Milk fresh 4 oz. or skim milk powder ¾ oz. or tinned milk 1 ⅓ oz or cheese ¾ oz. or egg No. 1.
Ghi "	...	1	1	1	2½	½	2	1	or Chicken dressed 2 oz. or Rabbit dressed 2 oz.
Bengers' food/Horlicks' oz.	...	...	...	...	...	...	...	...	(e) Chicken or rabbit dressed 10 oz. is equivalent to chicken or rabbit live weight 20 oz.
Butter/milk	1	...	...	...	...	...	...	...	
Butter/margarine	...	...	...	...	...	...	...	...	
vitaminised	—	...	...	½	½	...	...	...	
Meat, Poultry, fish (e)									
Mutton fresh incl. bone oz.	...	...	...	6	8	...	8	...	
Chicken or rabbit dressed	...	...	...	...	...	...	...	...	
(f) (oz.)	10	10	10	...	...	5	...	...	
Fish fresh oz.	...	...	...	...	...	4	1½	...	

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Liver	...	2	2	2	4	4	...	...	...	(f) Chicken dressed in diets "F" and "I" is included for making broth.
Vegetables and fruit.										(g) Where fruit fresh citrus is specified it is intended for preparing fruit drinks. If citrus fruits are not available, the medical officer may give ascorbic acid dissolved in lime juice cordial.
Fruit fresh citrus (g)	...	8	8	8	8	8	...	...	...	(h) In the case of 'O' diet 3½ oz. "Basin" (dal channa flour) should be supplied per head per week in lieu of an equal quantity of dhal.
Fruit fresh variety	...	...	...	8	...	...	8	8	...	(i) For every five pints of barley water 2 oz. Barley pearls and 2 oz. sugar are allowed. For every pint of fruit juice cordial, as many limes, lemons, or oranges as will produce 2 oz. of prepared fruit juice, and 1 oz. of sugar is allowed. Aerated water should only be ordered when certified as necessary by the officer in charge.
Vegetables fresh	...	...	...	8	...	...	8	8	...	(j) When R.I.A.S.C. condiment powder is supplied 25% of the powder be substituted by fresh condiment according to requirements.
Potatoes fresh	...	...	...	4	...	...	4	4	...	(k) See Note VI.
Onions fresh	...	...	...	1	1	1	2	2	1	
Dal	...	...	...	1	1	1	2	2	2(h)	
Sugar.										
Sugar	...	4	3½	3	3½	3	3	3	2	
Beverages.										
Tea	...	¼	¼	¼	¼	¼	¼	¼	¼	
Cocoa powder	...	...	On as	required	...	...	...	...	...	
Barley water (i)	...	...	"	"	"	"	"	"	"	
Fruit juice cordial	...	...	"	"	"	"	"	"	"	
Aerated water	...	...	On as	required	...	...	...	...	...	
Condiments.										
Condiments (j)	...	...	On as	required	...	...	...	...	...	
Achar	...	...	On as	required	...	...	...	...	...	
Miscellaneous.										
Salt	...	1	½	¾	¾	¾	¾	¾	¾	
Ice } Jelly }	...	...	On as	required	...	...	...	...	...	
Firewood (k) lbs.	...	3	3	3	3	3	3	3	2	

NOTES:—

(i) Subsistence diet will be drawn by patients admitted after 1200 hours for day of admission. Thereafter patients will be placed on one of the standard diets or on "No diet" with extras.

(ii) In cases in which patients cannot be placed on a diet, the words "No diet" will be entered on the diet sheet and they will be subsisted on extras.

(iii) Extras are not permissible with ordinary diets.

(iv) On "J" diet one teaspoon full of Sharkliver Oil or other vitamin A containing product should be administered daily.

(v) In diets "F", "R", "C", "J", "G", the scale of rations should be supplemented by 1½ oz. vegemite/marmite/yeast daily.

(vi) For each "No diet" and "Detained case" the scale of wood is 3 lbs. subject to the following rules which apply to all classes of diets:—

(a) When the number of patients (including those on "No diet") in each category for whom separate kitchens are maintained viz., Hindus, Mohammedans and others, is less than 12, an increased scale of 6 lbs. of firewood per patient may be issued separately up to the maximum of 36 lbs. if recommended by the O. C. hospital.

(b) There is no fixed limit of fuel for cooking purposes in the case of an outbreak of epidemic disease or other extraordinary occurrence.

(c) Oil, fuel, or charcoal, common, may be issued in lieu of firewood, provided the cost does not exceed that of the wood authorised.

NUTRITIONAL DISEASES IN INDIA

Hospital Diets and Extras.

Daily Scale of Extras for British and Indian Troops.

Note :—The undermentioned items will not be issued with ordinary diet.

*Where “B” or “I” is specified in column, the items referred to will be restricted to British or Indian patients respectively.

Extra.		* Maximum quantity that may be ordered in a day.	Substitute.	Remarks.
	1	2	3	4
1. Atta	...	I	12 ozs.	
2. Bacon	...	B	2 „	
3. Beef tea	...	B	1 pint.	... For each pint, 12 ozs. beef without bone or 15 ozs. beef with bone or $\frac{3}{4}$ ozs. meat extract are allowed.
4. Biscuit fancy	...		2 ozs.	
5. Bovril	...		...	
6. Bread	...		8 ozs.	
7. Butter	...		1 oz.	
8. Cheese	...		$\frac{3}{4}$ oz.	
9. Chicken broth	...		...	... 8 oz. chicken (dressed weight) for $\frac{3}{4}$ pint of broth.
10. Cocoa	...		1 pint.	... For each pint, cocoa $\frac{1}{4}$ oz., milk 2 ozs. sugar $\frac{1}{2}$ oz.
11. Coffee	...		...	Tea For each pint, coffee $\frac{1}{2}$ oz.; milk 2 ozs.; sugar $\frac{1}{2}$ oz.
12. Condiments	...	I	$\frac{1}{4}$ oz.	
13. Cornflour	...		...	Rice 1 oz. sugar for 2 ozs. cornflour or rice is allowed,
14. Cream fresh	...		2 ozs.	Cream tinned

Hospital Diets and Extras.

Daily Scale of Extras for British and Indian Troops—(continued).

Extra.		*	Maximum quantity that may be ordered in a day.	Substitute.	Remarks.
1	2	3	4	5	
15.	Dal	...	I 3 ozs.		
16.	Eggs	...	...		
17.	Fish fresh	...	8 ozs.		
18.	Food, Bengers	...	...	Any accepted dried milk food available	
19.	Fruit dried	...	2 ozs.	...	Almonds, raisins, sul- tana prunes.
20.	Fruit fresh	...	8 ozs.	Fruit tinned	
21.	Gelatins sheet	...	2 ozs.		
22.	Ghi	...	I 1 oz.		
23.	Groundnuts	...	1 oz.		
24.	Jam	...	1 oz.	Marmalade	
25.	Jelly, chicken or fruit...	...	...	...	For 8 ozs. chicken jelly:—chicken live weight 1 lb., eggs 2; sugar 1 oz.; stimu- lant if required.
26.	Lard	...	B 1 oz.	Dripping, mutton fat, suet.	
27.	Liver fresh	...	1 lb	Marmite 1 oz.	When required for treatment purposes.
28.	Macaroni	...			
29.	Milk fresh	...	60 ozs.	Milk tinned	
30.	Oatmeal	...	2 ozs.	...	10 ozs. milk allowed for 2 ozs. oatmeal.
31.	Onion	...	4 ozs.		

NUTRITIONAL DISEASES IN INDIA

Hospital Diets and Extras.

Daily Scale of Extras for British and Indian Troops—(continued).

Extra.		* Maximum quantity that may be ordered in a day.		Substitute.	Remarks.
1	2	3	4	5	
32. Potatoes	...	6 ozs.			
33. Rice, ration	...	2 ozs.			
34. Rice, water	...	...	...		For every 5 pints, 2 ozs. of rice and 2 ozs. sugar are allowed.
35. Sago	...	2 ozs.	Tapioca		
36. Sauces	...	...			
37. Sausages	...	B 3 ozs.			
38. Sugar	...	...			
39. Syrup Golden	...	1 oz.			
40. Tea	...	...	...		For each pint tea $\frac{1}{8}$ oz., milk 2 ozs., sugar 1 oz.
41. Vegetable fresh	...	6 ozs.	Vegetable tinned		
42. Vinegar	...	...			

APPENDIX 5

MENU OF A SPECIAL INVALID KITCHEN

All quantities are given on a scale for a single patient.

1. *Khir.*

Milk	...	...	...	8 ozs.
Rice	...	...	...	$\frac{1}{2}$ oz.
Sugar	...	...	...	$\frac{1}{2}$ oz.
Approximate calorific value	...	...	...	280

Preparation:—Wash and clean the rice: add the rice to the milk and cook on a slow fire, stirring all the while for half an hour. When ready, add the sugar. The total weight of *khir* will be 5-6 ozs.

2. *Egg Halwa.*

Eggs	...	...	...	2
Sugar	...	...	...	$\frac{1}{2}$ oz.
Milk	...	...	...	$\frac{1}{2}$ oz.
Ghee or butter	...	...	...	$\frac{1}{2}$ oz.
Approximate calorific value	...	...	...	390

Preparation:—Beat up the eggs and then add the sugar, milk and butter. Cook on a slow fire stirring all the while for about ten minutes. When ready, the total quantity will be about 4 ozs.

3. *Halwa Sujji or Atta.*

Atta or Sujji	...	...	...	1 oz.
Sugar	...	...	...	1 oz.
Ghee	...	...	...	1 oz.
Water	...	...	...	3 ozs.
Approximate calorific value	...	...	...	460

Preparation:—(a) Put the Ghee in a pan or karchi. When the Ghee comes to the boil, then add atta or sujji and brown it by stirring all the time.

(b) Mix the sugar with hot water.

(c) Add (b) to (a) and keep stirring all the time. Stirring is very important. In a short time (5 to 10 minutes) owing to evaporation of water it will become thick in consistency. Total quantity will be 5 ozs.

4. *Bread Pudding.*

Bread	...	...	...	1 OZ.
Egg	...	...	...	1
Butter	...	...	...	$\frac{1}{2}$ OZ.
Sugar	...	...	...	1 OZ.
Milk	...	...	...	4 OZS.
Approximate calorific value	...	...	...	500

Preparation:—Beat up the egg and then add sugar and milk to it. Then butter the bread and place it in a pudding dish and pour the milk mixture over it. Put the dish in an oven for $\frac{1}{2}$ hour.

Total quantity will be 6 ozs.

5. *Custard Pudding.*

Milk	...	...	...	8 OZS.
Egg	...	...	...	1
Sugar	...	...	...	1 OZ.
Approximate calorific value	...	...	...	390

Preparation:—Beat up the egg well and then add milk and sugar to it. Put the mixture in a pudding dish in the oven for 20-30 minutes. Any kind of essence or cardamoms may be added for flavour. Total quantity 6 ozs.

6. *Halwa Moong Dal. (Specially for Vegetarians).*

Moong Dal	...	...	...	1 OZ.
Sugar	...	...	...	1 OZ.
Ghee	...	...	...	1 OZ.
Water	...	...	...	2 OZS.
Approximate calorific value	...	...	...	460

Preparation:—Soak the Dal in water for 12 hours and then remove the husk and grind the wet dal fine. The ground dal is then fried in ghee till it becomes slightly brown. Then add the hot water and sugar. Keep stirring all the while for about 5-10 minutes.

Total quantity=3 to 4 ozs.

7. *Khichri.*

Rice	...	...	...	2 OZS.
Dal Moong split	...	...	...	$\frac{1}{2}$ OZ.
Ghee	...	...	...	$\frac{1}{2}$ OZ.
Approximate calorific value	...	...	...	370

Preparation:—Wash and clean the rice and split the dal separately. Then add the dal to boiling water (one lb.). When the dal is half cooked, add the rice and ghee and let all cook slowly for about half an hour.

Total quantity=12 to 14 ozs. or so.

8. *Chicken Curry or Korma.*

Chicken	...	...	...	8 ozs.
Ghee	...	...	...	1 oz.
Onions	...	...	...	1 oz.
Condiments	...	...	...	$\frac{1}{4}$ oz.
Approximate calorific value	...	...	...	500

Preparation:—Wash and clean the meat. Then fry the onions and condiments in ghee. Now add the chicken and fry for 10 minutes. Add hot water as required and cook on a slow fire for about 45 minutes.

9. *Vegetable Curry.*

Vegetables	...	...	...	8 ozs.
Ghee	...	...	...	$\frac{1}{2}$ oz.
Onion	...	...	...	$\frac{1}{2}$ oz.
Condiments	...	...	...	$\frac{1}{2}$ oz.
Approximate calorific value	...	...	...	210

Preparation:—Wash, peel and cut the vegetables. Fry the onions and condiments in ghee, till the onions get brown. Then add the vegetables and water if required, (depending on kind of vegetable used). Cook on a slow fire for ten to fifteen minutes.

10. *Fried Liver.*

Liver	...	...	...	4 ozs.
Ghee	...	...	...	$\frac{1}{2}$ oz.
Onions	...	...	...	$\frac{1}{2}$ oz.
Condiments	...	...	...	$\frac{1}{2}$ oz.
Approximate calorific value	...	...	...	295

Preparation:—Fry the onions and condiments in ghee. After washing and cutting the liver into small pieces, put the liver into a pan and cook on a slow fire for 2 to 5 minutes only.

11. *Fresh Cheese Curry. (Specially for Vegetarians).*

Milk	...	...	...	8 ozs.
Curd	...	...	...	1 oz.
Ghee	...	...	...	$\frac{1}{2}$ oz.
Onions	...	...	...	$\frac{1}{4}$ oz.
Condiments (salt, pepper, ground cardmonon, haldi)	...	...	...	$1/16$ oz.
Water	...	...	...	2 ozs.
Approximate calorific value	...	...	...	350

Preparation:—Boil the milk, add curd to it and keep it boiling for about five minutes. Then the whole will form a curd. Tie the curd in a piece of muslin and drain off the fluid. By this process the cheese will be separated in a mass. Cut the cheese into squares and fry the cheese in Ghee. Take the cheese out of the sanapan and then fry the onions and condiments in the same ghee. Now again add the cheese and some hot water. Let this all simmer for 5 to 10 minutes.

12. *Lasi.*

Curd	...	...	...	8 ozs.
Water	...	...	...	12 ozs.
Salt or sugar to taste.				
Approximate calorific value	...	...	...	250

Preparation:—Churn curd well for 10 to 15 minutes till the particles break up into a smooth paste. Add water and cool in the refrigerator, if possible.

13. *Fresh Liver Juice.*

Fresh Liver	...	...	...	2 ozs.
Lime Juice	...	...	...	$\frac{1}{4}$ oz.
Sugar	...	...	...	$\frac{1}{2}$ oz.
Water	...	...	...	1 oz.
Approximate calorific value	...	...	...	100

Preparation:—Mince the liver thoroughly. Add water and strain through a fine muslin. Add lime juice and sugar. Serve at once.

14. *Germinating Gram.*

Preparation:—Wash and clean the gram. Soak it in water for 24 hours. Then lay the gram flat between wet sheets or towels. Keep it moist for 12 hours. The gram will then start sprouting. It is now ready for issue. To make it more palatable sprinkle over it a little fresh lime juice, salt and pepper.

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